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ALLERGY TO ASPIRIN*

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ALLERGY to various simple drugs has been observed for a long time and widely reported. Many forms of allergic reaction are seen including urticaria, angio-œdema, drug eruptions, migraine, gastro-intestinal symptoms, hydrarthroses, agranulocytosis, rhinitis and asthma. Such drugs as arsphenamine, gold, amidopyrine, the sulfonamides, cincophen, phenolphthalein, barbiturates, cocaine, pontocaine, novocaine, quinine and aspirin have all been shown to be capable of producing severe allergic reactions.

As long ago as 1919, Cooke emphasized that typically allergic reactions occurred with many drugs in some patients, and pointed out that these allergic reactions were not to be confused with exaggerated normal reactions to the drug, *i.e.*, idiosyncrasy. In drug allergy a dramatic reaction occurs with very small amounts and produces typical allergic manifestations. Such reactions are not the normal or ordinary pharmacological behaviour of the drug and are similar to the reactions produced by totally unrelated substances such as foods, dusts, etc.

It is not the purpose of this communication to discuss the broad field of drug allergy, important as it is, but to consider one of the most common, and perhaps most serious of these allergies—that associated with aspirin.

Aspirin (acetylsalicylic acid) is perhaps the most widely used of all drugs. Frequently knowledge of its presence is obscured by trade names, and very often it is impossible for the layman to recognize it under its chemical name. Doctors often prescribe it in capsule form without the patient's knowledge. In relation to its very wide use the number of cases of aspirin sensitivity is small, but the effects are so striking and often dangerous that physicians should be familiar with its character.

There are many reports in the literature dealing with this subject. Urticaria and angio-œdema are common manifestations, as is rhinitis and asthma is a common and serious result of this sensitivity. Cooke, Prickman and Buchstein, Benson, Vaughan, Feinberg, Unger and many others have reported violent or sudden asthmatic death from aspirin. Many observers have drawn attention to the fact that aspirin sensitivity tends to occur in the most severe and intractable cases of asthma, often with non-reacting skin tests, always in adults and usually in the older age groups. It appears to be twice as frequent in women as in men. Various estimates of the frequency of aspirin sensitivity have been made, ranging from 2 to 10% of asthmatic patients. The occurrence of aspirin sensitivity in an asthmatic is often considered as a bad prognostic import.

The mechanism of aspirin and other simple drug sensitivity is not definitely known but the most widely accepted view is that of Landsteiner. The simple chemical substance is not an allergen *per se*, but it is believed that it combines with body proteins, perhaps the circulating globulins, to form specific allergens or haptens. The allergic reactions are produced by these combined substances.

It has been our experience that knowledge of drug allergy, particularly this most common and important one, aspirin sensitivity, is not widespread among the profession or the laity, and we felt that a report of some of our own experiences might serve to emphasize an important medical problem.

Aspirin and other drug sensitivity is more apt to become apparent in hospitalized patients because all medications are recorded and sudden changes in the patient's condition noted. Only the most intelligent and co-operative office patients will keep their doctor acquainted with all their medications, and few think it necessary to mention such a common thing as aspirin, even when they are keeping a meticulous food and

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symptom diary. There is then an obvious necessity to scrutinize carefully all medications which the patient is taking and for the doctor to be familiar with the aspirin content of a wide variety of proprietary and patent medicines.

A dramatic experience of ours may serve to emphasize the difficulty in ascertaining such data. An asthmatic woman 41 years of age, was under investigation in the office. In answer to routine enquiry she said she was unware of any unusual drug reactions and told us that she had been accustomed to take "Frosst 217" tablets for headache. As her asthma became more difficult to control she was admitted to hospital. With the usual measures she gradually improved and in several days became almost symptom-free and her chest signs disappeared. While making rounds one morning she complained to us of a headache and requested two tablets of "Frosst 217". I concurred and left the ward. Within a matter of minutes I was urgently recalled to her ward and found her in a state of collapse. She was deeply cyanosed, a few rhonchi could be heard but no other breath sounds, the blood pressure could not be read and she appeared to be desperately ill. Active resuscitation with oxygen, adrenalin, coramine and aminophyllin finally restored consciousness, a normal blood pressure and fairly good respirations with little dyspnœa or cyanosis.

This woman had never had such a severe reaction before but in retrospect it was realized that her normal cycle was headache, aspirin, asthma, adrenalin and headache. She later proved to be food and dust sensitive. She had nasal polypi and evidence of sinus disease. She died within a year of her discharge from hospital. As she resided in a distant town no further data were obtained.

The intern and nurses on her ward never forgot this dramatic and frightening experience and have since been cautious in their use of drugs on asthmatic patients without the full knowledge and written authority of the attending physician. However, it is notoriously difficult to prevent nurses and interns from administering such customary analgesics as aspirin and its compounds, believing them to be innocuous. We have felt it important to try to emphasize to our nursing and intern staff the dangers of such medications in asthmatics and always write an order forbidding the use of any medication without express authority.

We have reviewed 830 successive cases of asthma seen in the period January 1, 1946 to April 30, 1950. In this series we found 22 cases of definite aspirin sensitivity, *i.e.*, 2.7% of the whole. This is to be compared with reported incidences of from 2 to 10%. As more than a quarter of our 830 cases of asthma were in children and since none of these were aspirin-sensitive, the incidence of aspirin sensitivity in our adult group is nearer to 3.5%. Fifteen of the aspirin-sensitive cases were women. This ratio of two females to one male is almost identical with the various reported series.

The ages at which these patients were first seen were:

Age	*Cases*
20 to 29	4
30 to 39	4
40 to 49	6
50 to 59	7
60	1
	22

A prominent hereditary allergic history was found in 12 or 54%.

We attempted to grade the severity of asthma in our aspirin-sensitive cases as follows:

Grade	I	3 Cases	(mild)
Grade	II	8 "	(controlled by simple medications)
Grade	III	6 "	(requiring adrenalin, etc.)
Grade	IV	5 "	(very severe with frequent status)

It is comonly held that aspirin-sensitive cases are much more severe than the others. Only half of our cases could be classified as severe, that is grades III and IV. These 11 cases varied in age from 27 to 64 years and 9 of them were women. In the whole series of 830 cases, only 13% were classified as severe or in grades III and IV.

All five of our grade IV cases died during the four year period of study. That is, 22.7% of our aspirin-sensitive cases were dead within four years. Three of these cases were examined at autopsy and showed the characteristic findings of death from bronchial asthma.[1]

We think it is fair to say that our experience and that of others is that the presence of aspirin sensitivity has in general a serious prognostic significance. But half of our own aspirin cases were mild and have not changed materially, to our knowledge, in the four year period.

It is commonly reported that aspirin-sensitive cases, except those with pollinosis, are frequently insensitive to routine skin testing. In our group of 22 cases, five showed no skin reactions. Of the other 17, 12 reacted to pollen, 13 to mould

spores, 11 to house dust, 12 to animal dander and 5 to feathers. All tests were done intracutaneously. Although there were definite skin test reactions in 17 cases, in only 12 of these did we feel that a satisfactory etiological diagnosis had been made. All our aspirin cases showed periodicity throughout the year.

We think that it is highly probable that there are many undetected cases of aspirin sensitivity in the whole series. The difficulty of detecting it in the absence of violent reactions is obvious. It has been reported and it is our experience that aspirin sensitivity may be mild and hence undetected until the sensitivity becomes more severe, or until it is demonstrated fortuitously under controlled conditions. One of our small series, Mrs. H., suffered from rheumatoid arthritis and was given a capsule containing aspirin. She had a very severe exacerbation of her asthma each time she took one of these capsules and after the third one would not try them again. She had no previous suspicion of drug sensitivity. Another woman, Mrs. W., was unaware of aspirin sensitivity until she was admitted to hospital for a minor surgical procedure and developed her most severe attack of asthma within a few minutes of being given aspirin as an analgesic.

In addition to the manifestation of asthma, 3 of these cases developed urticaria following the drug and one of them the symptoms of allergic rhinitis. Three of the aspirin cases also developed asthma after the administration of benadryl, pyribenzamine and antistine, respectively. One of the aspirin-sensitive cases, Mrs. G., was given carbrital (Parke-Davis) and developed severe angio-œdema of her palate, pharynx and larynx, but without signs of asthma. She recovered following repeated injections of epinephrine.

We did not attempt a skin test with aspirin or the other drugs because it is reported to be negative in most cases and is sometimes very dangerous.

DISCUSSION

The most common drug causing severe allergic reactions is aspirin. The most frequent symptom is asthma, but urticaria, angio-œdema and rhinitis are fairly common. In a series of 830 asthmatics we have discovered 22 cases of definite aspirin sensitivity. All of these cases manifested asthma with very small amounts of aspirin. Several of them also had urticaria, angio-œdema and rhinitis. In addition 3 of them were allergic to antihistamine drugs. All of the cases were in adults ranging from 27 to 64 years of age. Half of them had a severe grade of asthma as compared to only 13% of the whole series. Five of the 22 cases died in the four year period under study. Clinical reactions to aspirin are usually violent and rapid.

There is evidence that the key chemical grouping in aspirin is the acetyl radical and it can be shown that aspirin-sensitive patients may tolerate sodium salicylate.

Such small quantities of the drug are required to produce tremendous reactions that one naturally wonders if those intractable cases so common in our practice in whom a satisfactory etiology cannot be found are not also sensitive to some other trace chemical occurring in their daily dietary intake, as suggested by Feinberg.[2] Aspirin will not give a positive skin test and perhaps such a postulated trace chemical would not do so either. A simple common substance such as aspirin is often missed, we are sure, in the diagnostic survey and it is quite conceivable that some other common substance may also act similarly. So far as we are aware this suggestion of Feinberg's has not been carried further.

Aspirin sensitivity is highly dangerous and must always be looked for in the history. Skin-testing for it is useless and dangerous as pointed out by Cooke.[3] Trial of its oral administration should never be attempted if there is any reason to believe that hypersensitivity to aspirin exists.

Aspirin sensitivity generally occurs in the most serious cases of asthma and is therefore of some prognostic importance. The use of the drug may be fatal. However, it has long been known that aspirin may act beneficially in some cases of asthma and the drug has been strongly recommended for therapeutic use by such experienced clinicians as Duke. Certainly many asthmatics have found for themselves that aspirin helps them and I think the drug probably occurs in some patent asthma remedies.

"An open foe may prove a curse,
But a pretended friend is worse."
Gay.

SUMMARY

Twenty-two cases of aspirin sensitivity were discovered in a series of 830 successive cases of asthma. Five of these died within one year. All cases were adult and fifteen were women.

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ALLERGIE A L'ASPIRINE

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DEPUIS longtemps on a observé et rapporté des cas d'allergie à divers médicaments ordinaires. Il existe un grand nombre de réactions allergiques, entre autres l'urticaire, l'angio-œdème, les dermatites médicamenteuses, les troubles gastro-intestinaux, les hydarthroses, l'agranulocytose, la rhinite et l'asthme. Parmi les médicaments qui peuvent provoquer des réactions allergiques graves, mentionnons l'arsphénamine, l'or, l'amidopyrine, les sulfamidés, le cincophène, le phénolphtaléine, les barbiturates, la cocaïne, la pontocaïne, la novocaïne, la quinine et l'aspirine.

Déjà en 1919, Cooke soulignait chez quelques malades des réactions allergiques typiques provoquées par maints remèdes, manifestations qu'il importait de ne pas confondre avec les réactions exagérées à une drogue, c'est-à-dire l'idiosyncrasie. Dans l'allergie médicamenteuse des quantités infinitésimales déclanchent des réactions violentes, voire dramatiques, qui outrepassent les effets pharmacologiques normaux de la drogue, ressemblant ainsi à ces manifestations que provoquent des substances qui n'ont aucune relation avec elle, à savoir des aliments, des poussières, etc. Aussi importante que puisse être la vaste question des allergies médicamenteuses, une seule d'entre elles fera l'objet de cette étude, celle de l'aspirine, qui est l'une des plus fréquentes et vraisemblablement aussi l'une des plus graves.

L'aspirine (acide acétylsalicylique) est sans contredit le médicament qui est le plus universellement employé, et cependant peu fréquents sont les cas de sensibilité à cette drogue, mais il importe au médecin d'en être bien averti à cause du caractère remarquable et souvent dangereux des accidents qu'elle provoque. Il arrive souvent au patient d'en prendre sans le savoir, camouflée qu'elle est sous de nombreuses marques de commerce; le médecin lui même la prescrira sous forme de capsule à l'insu de son malade.

Le sujet en question abonde dans la littérature médicale. De même que la rhinite, l'urticaire et l'angio-œdème sont des manifestations communes, ainsi est l'asthme qui prend souvent une forme grave. Cooke, Prickman et Buchstein, Benson, Vaughn, Feinberg, Unger et bien d'autres encore ont publié des observations de morts subites dues à l'asthme et causées par l'aspirine. Ils soulignèrent, toujours chez des adultes plutôt avancés en âge, et deux fois plus fréquemment chez la femme que chez l'homme, une tendance à la sensibilité à l'aspirine dans les cas d'asthme les plus graves et les plus rebelles, souvent avec des épreuves cutanées négatives. Il a été estimé de 2 à 10% la proportion d'asthmatiques qui etaient sensibles à l'aspirine, et cette particularité comporte aouvent un fâcheux pronostic.

Landsteiner explique ainsi le mécanisme de la sensibilité à l'aspirine et à d'autres drogues courantes, et c'est son avis, sur ce point encore obscur, qui rallie la plupart des suffrages. La simple substance chimique n'est pas un allergène *per se*, mais elle se combinerait avec des protéines de l'organisme, des globulines du sang peut-être, et c'est cette combinaison qui provoquerait des réactions allergiques.

Ni le médecin ni le profane, croyons-nous par expérience, ne sont pas très au courant de ce phénomène d'allergie médicamenteuse, notamment celle de l'aspirine, la plus importante et la plus commune de toutes; il y a là un problème médical d'envergure qui pourrait être éclairé à la lueur de quelques-unes de nos propres expériences.

Il est plus facile de se rendre compte de la sensibilité à l'aspirine ou à toute autre drogue sur des malades hospitalisés parce qu'on prend note des médicaments qu'ils prennent ainsi que des changements soudains qu'ils produisent. En visite au bureau du médecin seuls les patients les plus éveillés et mieux intentionnés le mettront au courant de tous les remèdes qu'ils prennent, en omettant cependant pour la plupart de faire mention d'une drogue aussi banale que l'aspirine, même s'ils prennent rigoureusement note de leur alimentation ou de leurs symptômes. Le médecin se devra donc de toute nécessité de faire une enquête fouillée sur les médicaments que prend son malade, en plus de bien connaître la teneur en aspirine d'un très grand nombre de spécialités pharmaceutiques et de remèdes brevetés.

L'histoire dramatique suivante souligne bien la difficulté qu'il peut y avoir d'obtenir de tels renseignements. Une asthmatique âgée de 41 ans qui vint me consulter à mon cabinet me répondit en interrogatoire qu'elle n'avait jamais ressenti de réactions médicamenteuses inusitées, ajoutant qu'elle avait l'habitude de prendre des comprimés "Frosst 217" pour ses maux de tête. Son asthme empirant, elle fut admise à l'hôpital. La thérapie habituelle en ces cas aidant, son état s'améliora peu à peu, ses symptômes s'amendèrent en quelques jours avec disparition des signes pulmonaires. Au cours d'une de mes visites un matin, elle se plaignit de céphalée et réclama deux comprimés de "Frosst 217". J'acquiesçai et quittai les lieux. A peine quelques minutes après, je fus mandé d'urgence à sa salle et la trouvai en état de collapsus. Fortement cyanosée, le pouls filiforme, son état paraissait désespéré; à l'auscultation, quelques râles sibilants, rien d'autre. Après ranimation vigoureuse à l'oxygène, l'adrénaline, la coramine et l'aminophylline, la malade reprit conscience, la tension artérielle devint normale et les mouvements respiratoires reprirent avec peu de dyspnée ou de cyanose.

Cette femme n'avait jamais éprouvé une réaction aussi grave auparavant, mais un interrogatoire rétrospectif révéla que son cycle normal consistait en céphalée, aspirine, asthme, adrénaline, céphalée. Plus tard on apprit qu'elle était sensibilisée aux aliments et aux poussières, en plus de souffrir d'un polype nasal et d'une sinusite confirmée. Elle mourut dans l'année qui suivit sa sortie de l'hôpital, et comme elle habitait une ville éloignée on n'en sut pas plus long sur son cas.

L'interne et les infirmières alors en devoir dans sa salle n'oublièrent pas de sitôt cette tragique et effrayante affaire et depuis ce temps n'osent plus donner de médicaments à leurs malades asthmatiques à l'insu du médecin de service et sans son autorisation écrite. C'est à cette condition seule qu'ils éviteront des incidents regrettables, tellement courante est cette pratique, chez le personnel médical étudiant, de donner des analgésiques communs comme l'aspirine et ses composés parce qu'ils les croient inoffensifs.

Nous avons passé en revue 830 cas successifs d'asthme vus du 1 janvier 1946 au 30 avril 1950. De ce nombre il s'est trouvé 22 cas de sensibilité nette à l'aspirine, soit une proportion de 2.7%, comparable aux taux rapportés variant de 2 à 10%. Etant donné que plus que le quart de nos 830 cas d'asthme comprenait des enfants dont pas un seul n'était allergique à l'aspirine, le taux de sensibilité à cette drogue dans notre groupe d'adultes se rapproche davantage de 3.5%. Dans ce groupe on compte quinze femmes; ici encore la proportion de deux femmes pour un homme s'affirme.

Les âges de ces malades lors de leur premier examen étaient comme suit:

Age	*Cas*
20 à 29	4
30 à 39	4
40 à 49	6
50 à 59	7
60	1
	22

Dans 12 cas, soit une proportion de 54%, on trouva des antécédents nets d'hérédité allergique.

Nous tentâmes de fixer de la façon suivante le degré de gravité de l'asthme chez nos allergiques à l'aspirine:

Degré I 3 cas (bénins)
Degré II 8 cas (amendés par de simples médications)
Degré III 6 cas (nécessitant de l'adrénaline, etc.)
Degré IV 5 cas (très graves)

On est communément d'avis que les malades allergiques à l'aspirine sont beaucoup plus gravement atteints que les autres. Seuls la moitié de nos cas ceux des degrés III et IV, peuvent être classés comme graves. Ces onze malades, dont neuf femmes, étaient âgés de 27 à 64 ans. Dans la série entière de 830 cas, une proportion de 13% seulement était gravement malade ou dans les degrés III et IV.

Tous les cinq patients du degré V moururent au cours des quatre années que dura l'étude, c'est-à-dire 22.7% de nos cas allergiques à l'aspirine. Trois d'entre eux furent examinés à l'autopsie et montrèrent les signes caractéristiques de la mort par asthme bronchique.

En nous basant sur notre expérience et celle des autres, nous croyons devoir affirmer que l'allergie à l'aspirine assombrit considérablement le pronostic. Cependant la moitié de nos cas d'aspirine étaient bénins, et ils n'ont guère changé au cours de ces quatre années.

On est d'accord pour dire que les cas d'allergie à l'aspirine, sauf ceux qui souffrent aussi de la fièvre des foins, ne réagissent guère aux tests cutanés de routine. Ce fut le cas pour cinq de notre groupe de 22 malades. Parmi les

autres 17, 12 réagissaient au pollen, 13 aux spores de moisissures, 11 à la poussière des maisons, 12 au poil d'animaux et 5 aux plumes. Tous ces tests furent pratiqués par voie dermique. En regard de 17 cas positifs, 12 seulement nous permirent de porter un diagnostic satisfaisant.

Parce qu'il n'est pas facile, en l'absence de réactions caractéristiques, de déceler les cas réels d'allergie à l'aspirine, il est fort probable qu'il nous en soit échappé un bon nombre de notre série entière. Notre expérience, confirmée par celle d'autres chercheurs, nous a enseigné que cette allergie à l'aspirine peut être légère et passer inaperçue jusqu'au moment où sa gravité s'affirme ou qu'elle soit l'effet du hasard. Ainsi, Mme H., qui souffrait d'arthrite rhumatoïde, n'avait jamais jusqu'ici présenté d'allergie à la drogue. Après avoir pris une capsule contenant de l'aspirine, elle vit son asthme s'exacerber de sérieuse façon chaque fois, et refusa désormais d'en prendre aprés une troisième crise. Une autre femme, Mme W. ne se sut allergique qu'après avoir été hospitalisée pour une intervention de petite chirurgie, et elle fit sa plus grave crise d'asthme quelques minutes à peine après avoir absorbé de l'aspirine comme analgésique.

En outre de manifestations asthmatiques, 3 malades eurent des crises d'urticaire et l'un d'eux, une rhinite allergique. Trois autres cas d'aspirine firent de l'asthme après ingestion de benadryl, de pyribenzamine et d'antistine respectivement. Une autre malade, Mme G., à qui l'on donna du carbrital (Parke-Davis), contracta un angio-œdème du palais, du larynx et du pharynx, mais des injections répétées d'épinéphrine la ramenèrent à la santé.

Parce qu'il est habituellement négatif et parfois même dangereux, nous ne tentâmes pas de pratiquer un test cutané avec l'aspirine ou ses composés.

DISCUSSION

L'aspirine est le médicament le plus en usage qui provoque de graves réactions allergiques. L'asthme en est le symptôme le plus fréquent, encore que l'urticaire, l'angio-œdème et la rhinite soient assez communs. Dans une série de 830 asthmatiques nous découvrîmes 22 cas d'allergie nette à l'aspirine. Des crises d'asthme furent déclanchées chez tous ces malades avec de très petites doses d'aspirine. Plusieurs d'entre eux présentèrent également de l'urticaire, de l'angio-œdème et de la rhinite, et parmi ceux-là trois se montrèrent allergiques aux produits antihistaminiques. Tous ces patients étaient des adultes dont les âges variaient de 27 à 64 ans, dont la moitié firent une crise grave d'asthme. De ces 22 cas cinq moururent au cours des quatre années qu'ils furent sous observation. Les réactions cliniques à l'aspirine sont habituellement violentes et rapides.

Il suffit de si petites quantités de la drogue pour provoquer des réactions formidables que l'on est en droit de se demander si ces cas rebelles fréquents en pratique et dont l'étiologie nous échappe ne sont pas également allergiques à quelque autre substance chimique que l'on trouverait dans le régime alimentaire quotidien. Tel est l'avis de Feinberg. L'aspirine ne donnera pas un test cutané positif, ni peut être cette autre substance chimique non plus, et il n'y a rien d'étonnant qu'il n'en soit pas fait mention au moment du diagnostic. Jusqu'ici nous ne croyons que la suggestion de Feinberg ait fait beaucoup de chemin.

L'allergie à l'aspirine est très dangereuse, aussi faut-il toujours la rechercher dans l'histoire d'un cas. Cooke a souligné l'inutilité et le danger des tests cutanés pour la déceler. On ne devrait donc jamais prescrire l'aspirine s'il y avait raison de penser que la malade y est hypersensible.

L'allergie à l'aspirine survient généralement dans les cas d'asthme les plus graves, elle a donc une valeur pronostique certaine. L'emploi du médicament peut être fatal. C'est un fait depuis longtemps acquis, cependant, que l'aspirine est utile dans certains cas d'asthme, et des cliniciens réputés tels que Duke en recommandent fortement l'usage. Il n'y a aucun doute que nombre d'asthmatiques s'en sont bien trouvés, et c'est mon avis que certains remèdes brevetés contre l'asthme contiennent l'aspirine.

"Que le Seigneur me protège contre mes amis;
quant à mes ennemis, je m'en charge."
(Fontenelle)

RÉSUMÉ

Vingt-deux cas d'allergie à l'aspirine furent mis à jour dans une série de 830 cas successifs d'asthme. Cinq d'entre eux moururent en moins d'un an. Tous les malades étaient des adultes, parmi lesquels on comptait quinze femmes.

RHEUMATIC FEVER TREATED WITH CORTISONE AND ACTH*

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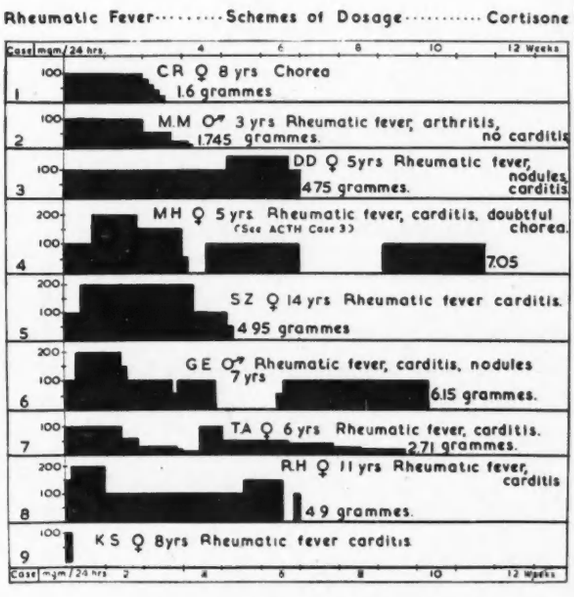
IN May, 1949, Hench and co-workers reported 3 cases of rheumatic fever treated with cortisone.† In May, 1950, Massell *et al.* recorded their findings in 10 cases treated with ACTH.† Since then a few additional cases have appeared in the literature. In most instances the published reports indicate beneficial effects of these hormones in rheumatic fever patients. During 1950 we have treated 23 cases of rheumatic fever with either cortisone or ACTH and the results are reported below. Three of the cases are still receiving treatment, and the others have been followed for between 1 and 4 months since stopping treatment.

Procedure: (see Figs. 1 and 2) (the charts include only the first 18 cases to receive hormone therapy).



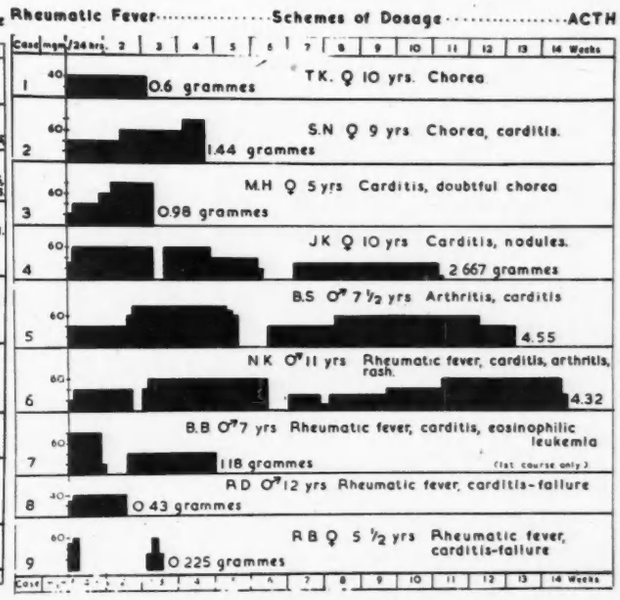


Fig. 1.—Showing quantity and duration of therapy in first 9 consecutive cases on cortisone. N.B. The case number, given in the left margin, is used in all subsequent cases.
Fig. 2.—Showing quantity and duration of therapy in first 9 consecutive cases on ACTH.

Cortisone was administered twice daily. The total daily dose was 100 to 200 mgm. in 24 hours. When ACTH was administered, 40 to 80 mgm. were given in 24 hours, depending on the size of the child. It was given in 4 hourly doses. Such therapy was continued until signs of active disease had subsided and the rheumatic fever remained quiescent without the suppressing effects of hormone therapy. Most children were treated 4 to 6 weeks and then the injections stopped. When evidence of a relapse occurred the treatment was restarted and kept up for 2 to 3 weeks more. After a second course any reappearance of activity was usually transient.

Twenty-three cases of acute rheumatic fever admitted to the Hospital for Sick Children in recent months have been treated with one or the other of these hormones: 11 have been treated with cortisone only; 11 with ACTH only and one with ACTH followed by cortisone. The ages of the children treated varied from 3 to 14 years (average 7.7 years). There were 12 females and 11 males. Most cases were in their primary attack of rheumatic fever but 4 had a history of previous attacks and 2 had a history suggesting possible previous attacks; while one had established mitral stenosis and insufficiency, with no definite history of a clinical attack. The criteria for diagnosis were those set down by Jones in 1944.

SUMMARY OF RESULTS

Fever.—Fever was present in 12 cases at the onset of therapy. We were able to study this

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† The Cortisone and ACTH used in this study were provided by the National Research Council of Canada.

point on many more occasions since elevated temperatures recurred frequently when the hormone was temporarily discontinued. The temperature returned to normal in 1 to 4 days in all cases on cortisone; average 1.8 days. The children treated with ACTH had normal temperatures in 24 hours, except for 2 children who took 3 days and 8 days respectively. There appeared to be no difficulty in producing a normal temperature in these cases and it was usually not necessary to increase the amount of hormone in order to control temperature.

Arthritis.—Arthritis had subsided in all but 5 cases by the time treatment had begun: 3 of these lost all signs of pain and swelling within 24 hours from the onset of therapy, and the other 2 within 48 hours. Massell, using ACTH, reported clearing of arthritis within 1 to 2 days. Hench indicated that the arthritis disappeared within 1 to 3 days with cortisone.

Pericardial friction rub.—There were 3 cases with acute pericardial friction rub. One died within 12 hours of starting treatment, in one case the friction rub disappeared in 2 days, and in the third a friction rub lasted for 14 days and an effusion developed. Massell reports 4 children with pericardial friction rubs, and in 2 the rub disappeared in 1 to 3 days and in the other 2 it persisted for 11 to 14 days in spite of continued therapy.

Nodules.—There were subcutaneous nodules in 3 of our cases. The nodules disappeared in approximately the same time in each case, 19 to 22 days; that is, approximately 3 weeks from the beginning of therapy. In 2 cases, the nodules had appeared just as treatment was about to start; in the third case, the nodules had been present for 6 weeks before the onset of therapy. The general experience in the past has been that the nodules last from 1 to 6 months. They rarely disappear in less than 1 month. A sample was excised from each of these patients at various stages during therapy. Nodules removed at the start of treatment were typical of the disease; those removed toward the end of 3 weeks' therapy were much smaller, more difficult to locate under the microscope and showed microscopic evidence of healing.

Heart failure.—There were 8 cases with heart failure: 4 of these were the chronic type and 4 the acute. From our experience and from that reported in the literature, it is important to consider these 2 groups separately since there is a marked difference in the response to therapy. The 4 children with acute failure were in the early stages of rheumatic fever and were severely and desperately ill. One of these patients died within 12 hours of the first injection of cortisone and before we had a chance to treat her adequately. The second and third responded very rapidly and within a few days of starting ACTH were dramatically improved. The fourth case is still on therapy, which has only had a moderate clinical effect. Of the 4 children with chronic heart failure, 3 were receiving digoxin as well as hormone therapy. These cases improved slowly, but probably at the rate one might expect a case of chronic heart failure to improve with bed rest, digoxin and a low sodium diet. At times the administration of hormone therapy caused excessive gain in weight and an increase in their failure, and had to be stopped temporarily. The 4th case was bordering on failure on admission to hospital, and had had active rheumatic fever for a year and a half; after 3 weeks of hormone therapy she went into full-blown failure, thus making it necessary to stop the cortisone; digoxin was then started and the failure rapidly disappeared. A prolonged study of hormone therapy will be necessary before one can evaluate adequately the effects of such treatment in rheumatic heart disease with failure.

Chorea.—There were 4 cases of chorea in our study group and they all showed a steady clearing of this manifestation while on therapy, with disappearance of abnormal movements of hands and face within 3 weeks. (One case had slight residual weakness and incoordination for 6 weeks.) It may be of some significance that they all improved at the same rate and in the same time. In reviewing the course of 150 cases of chorea prior to 1930, Sutton found that signs cleared in an average of 42 days, mild cases clearing in an average of 27 days. Thus in this study of chorea cases appeared to become quiescent more rapidly than one would have expected without such therapy.

Gallop rhythm.—Eleven of the 23 cases had a third heart sound, at or inside the apex that we have classed as being gallop rhythm. In 4 of these cases the third sound disappeared in 4 weeks and in 6 it persisted throughout the treatment and stay in hospital. These latter

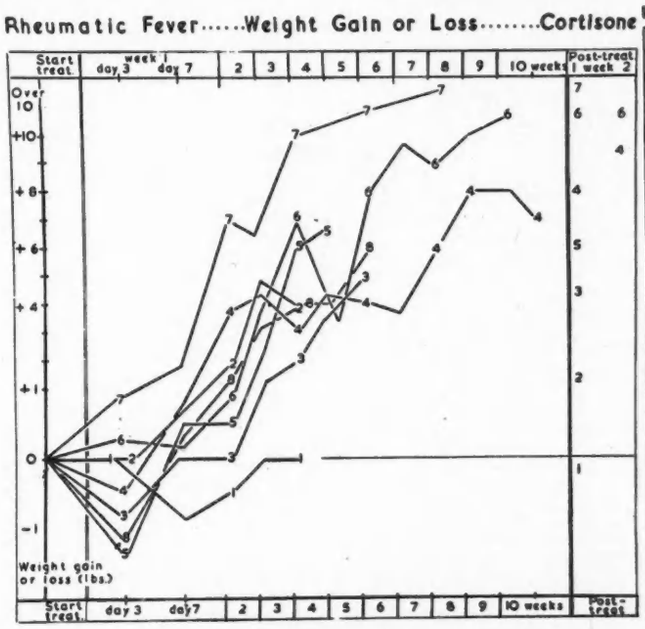
6 cases were all in failure and did not show the hoped-for response to treatment.

Murmurs.—There were 18 patients with mitral systolic murmurs; in 3 instances the murmurs became fainter; one very markedly so, but in no case did the murmur disappear. Three cases had aortic diastolic murmurs and these remained unchanged. Three had mitral diastolic murmurs which remained the same. In this connection it is interesting to note that Massell found that the murmurs disappeared in 2 of his cases treated with ACTH.

Heart size.—X-rays of the heart taken at weekly intervals showed that in some cases the heart got smaller and in others larger while under the effect of hormone therapy. The change in size by months has been set down in Fig. 7, and reveals considerable variation. In 2 cases there was a fairly marked decrease in size over a short period.

In 1942, Keith and Brick studied changes in heart size in rheumatic heart disease during the acute stages of the illness. To refresh our memories regarding the speed at which hearts returned to normal size in previous years among certain rheumatic children treated with salicylates, we reviewed the series previously reported. The x-rays of 3 selected cases are reproduced here (Fig. 8). In these 3 children, the hearts decreased in size from marked enlargement to approximately normal in 2 to 4 weeks. It was found that this rapid change in heart size occurred in approximately 10% of the children with acute rheumatic heart disease.

In reviewing the x-rays of 52 cases of rheumatic carditis in the first 3 months in hospital, during the years 1940-41, it was noted that 20 hearts became smaller; 10 got larger, and 22 showed no significant change. When they are charted according to change in heart size by month, they appear as shown in Fig. 7. They show somewhat the same scatter as the cases treated with ACTH and cortisone. The 2 groups however, are not comparable because they were investigated in different years and there were only half as many among the hormone-treated group as those treated with salicylates. However, they do show that considerably more study is necessary before one can draw clear-cut conclusions of the effect of ACTH and cortisone on the heart size in acute rheumatic fever.



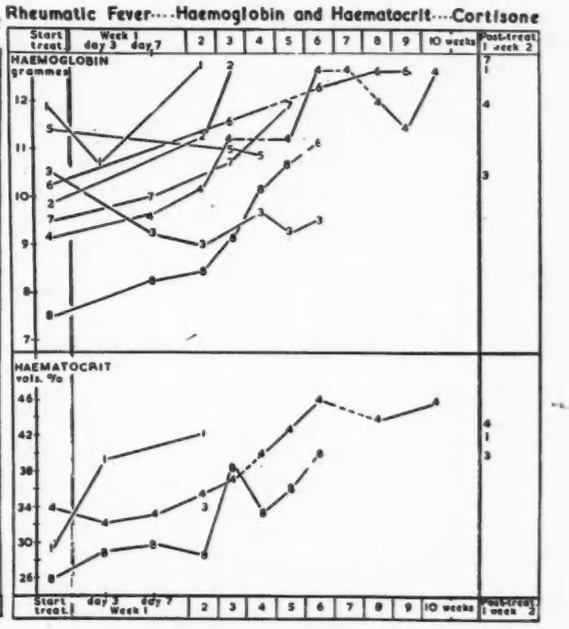


Fig. 3.—Showing weekly weight gains (pounds) in 8 cortisone treated cases. (Numerals = case numbers). **Fig. 4.**—Hæmoglobin levels at weekly intervals in 8 cortisone treated cases and hæmatocrit readings in 3, showing tendency to rise with prolonged therapy. (Broken lines represent stoppage of therapy temporarily).

Blood pressure.—Blood pressure was taken daily in all cases during treatment and no significant change was noted in any patient. There were merely the expected daily fluctuations.

Weight.—There were 18 children who showed a gain in weight during their period of hormone therapy. In 1 child there was no change in weight during treatment and in 3 there was a

loss in weight.

Among the 18 children who put on weight there was an average gain of 7.4 lb. (range 1 to 18 lb.). Among the children who lost weight there was an average decrease of 1¾ lb. (range ½ to 2½ lb.). Those that failed to gain or showed slight decrease in weight were those who were treated for the shorter intervals. The weight gains of some of the cortisone group are shown on Fig. 3.

Diuresis.—Five cases had a diuresis while still on hormone therapy and 10 had a diuresis after the hormone had stopped, which was sufficient to produce a significant drop in weight. Three children showed a diuresis both during and following the hormone therapy.

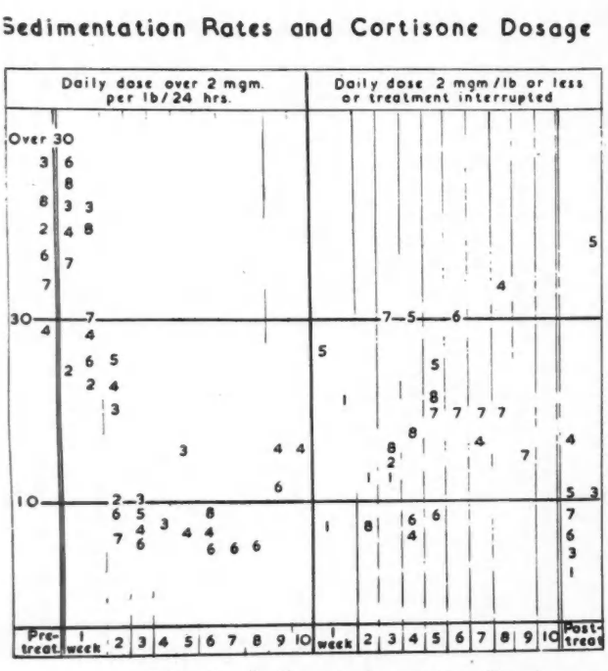
LABORATORY CHANGES

Sedimentation rate.—(The Landau method was used (see Fig. 5).

From past experience the sedimentation rate usually takes 4 to 6 weeks to return to normal but may remain up for many months or occasionally a year or more with an attack of rheumatic fever.

There is no doubt that the sedimentation rate returns to normal more rapidly with ACTH and cortisone, but when the hormone therapy is stopped after 4 to 6 weeks, there is frequently a rebound elevation of the sedimentation rate that makes the total span of increased sedimentation time approximately the same as is noted in children treated with salicylates.

As can be seen from Fig. 5, the sedimentation rate tended to be under 10 mm. in 1 hour more frequently in children receiving 2 mgm. per lb. per 24 hours of cortisone rather than in those on a smaller or interrupted dose. Among the ACTH group, the relationship between dose and sedimentation rate was less marked, but



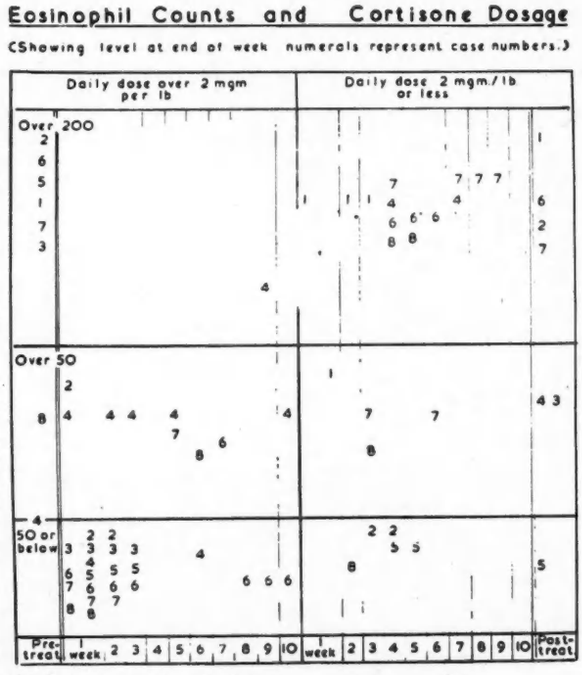


Fig. 5.—Sedimentation rates (Landau method), at onset of therapy and at weekly intervals, in first 8 cortisone-treated cases: showing the sedimentation rate tended to be lower during weeks when dose averaged over 2 mgm./lb./24 hours. (Numerals = case numbers). **Fig. 6.**—Eosinophile counts in same cases as Fig. 5, showing tendency to vary inversely with dosage of cortisone. (Numerals = case numbers).

The normal level was reached in 1 to 3 weeks in most cases. Subsequently when the hormone was stopped or the dose reduced, the sedimentation rate rose again; therapy was then restarted. The length of time from the beginning of treatment until the normal level of sedimentation rate was reached without the suppressing effect of the hormone was an average of 35 days (range 4 to 8 weeks).

these cases also showed a rise when the treatment was interrupted.

Eosinophiles.—(The eosinophiles were counted by an adaptation of the Dungar method).

The eosinophiles showed a marked drop when hormone therapy was begun in all but one case, but this depression frequently only lasted for about the first week of therapy (see Fig. 6). This was true for both cortisone and ACTH,

although the patients on ACTH showed a greater and more sustained depression of the eosinophiles than those on cortisone. Those receiving 2 mgm. cortisone per pound body weight daily, showed a greater drop than those on a smaller or interrupted dose. Taking both groups together whether treated by one hormone or the other, there was a drop in the eosinophiles to less than 50 per c.mm. in 14 cases (1 case had less than 50 eosinophiles per c.mm. on admission).

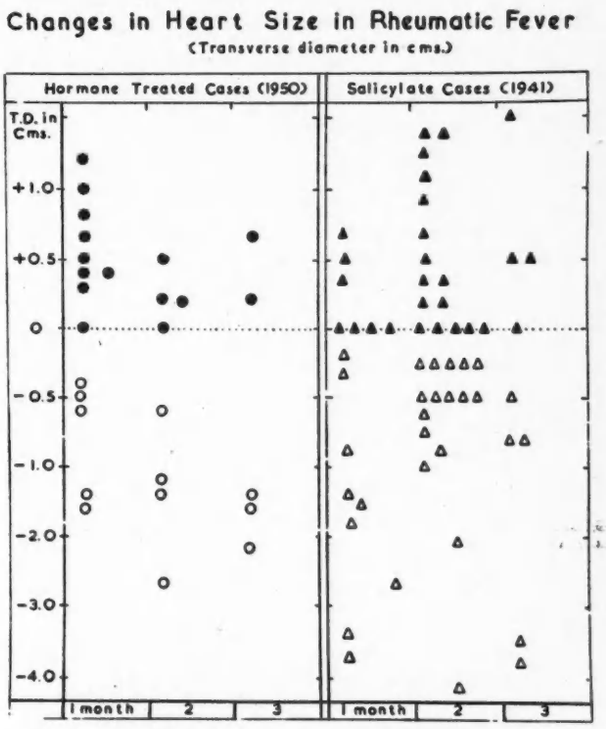


Fig. 7.—Changes in transverse diameter of heart (6 foot plates) as measured at monthly intervals from onset of therapy. Comparison of hormone treated cases with a group of salicylate treated cases. Showing that in both groups approximately equal numbers of cases enlarge or diminish in size.

The other 3 cases of the 17 had an eosinophile count which fell to between 1 and 200 eosinophiles per c.mm. As can be seen from the chart, one cortisone case showed a marked eosinophile escape towards the end of therapy; one of the ACTH group, although on a dose of 60 mgm. per day, developed a count of over 1,000 per c.mm. towards the end of therapy.

Hæmoglobin. — The hæmoglobin showed a significant rise in the majority of the patients (see Fig. 4). The first 11 cases treated showed an average increase of 1.8 gm. of hæmoglobin during therapy (range 0 to 5.2 gm.); 4 showed a drop in hæmoglobin averaging 0.8 gm. (range 0.3 to 1.7 gm.).

Hæmatocrit.—There was an increase in the hæmatocrit in all 15 cases studied at intervals during hormone therapy, average rise of 6.6 vol. % (range 2 to 15 vol. %).

OTHER EFFECTS OF HORMONE THERAPY

Several children developed signs of hyperadrenalism. Of the 20 cases who received hormone therapy for 2 weeks or more, 3 cases showed no change. As might be expected these were the 3 that were treated for the shorter period. Fifteen children developed side effects

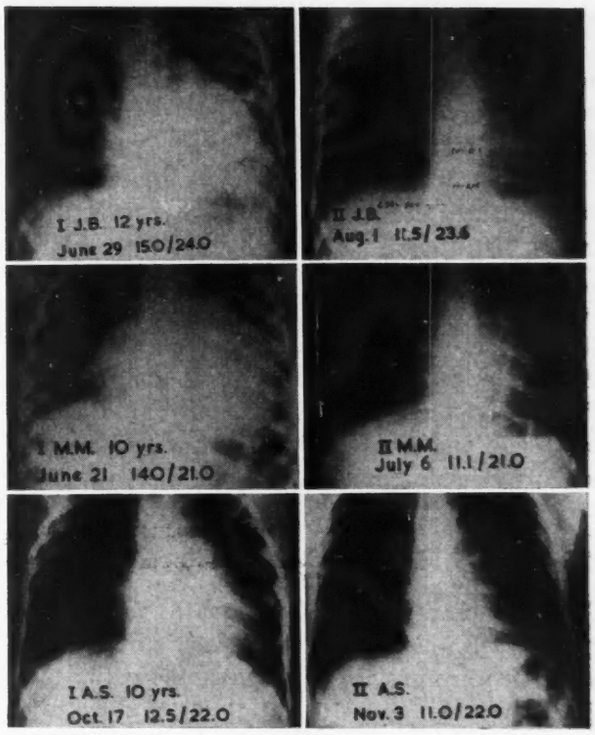


Fig. 8.—Above are shown the x-rays of 3 selected cases of acute rheumatic heart disease, treated with salicylates in 1940. The first x-ray in each case was taken on admission to hospital; the second x-ray two to four weeks later. Marked decrease in heart size is shown in each case over a relatively short interval.

listed as follows: facial rounding and some prominence of the abdomen in 15 cases: hirsutism in 6 cases: acne in 4 cases: abdominal pain in 3 cases: striæ in one. All 15 children felt better on therapy and in 4 there appeared to be some degree of euphoria. One child developed purpuric lesions on the skin of the abdomen and legs; these disappeared when the therapy was stopped.

The facial rounding and hirsutism disappeared after therapy was discontinued, but the regression took place slowly. In several cases these signs lasted 2 to 3 months after cessation of treatment.

Dosage.—A total daily dose of 40 to 60 mgm. of ACTH controlled the fever and arthritis effectively in these children between 5 to 13

with rheumatic fever; 100 mgm. daily of cortisone was usually sufficient to achieve the same purpose. The dosage had to be reduced or eliminated when signs of heart failure supervened.

We attempted to evaluate dosage on the basis of eosinophile response with either hormone: 1 mgm. per pound of ACTH produced roughly the same degree of depression of the eosinophiles as did 2 mgm. per pound of cortisone. The suppressing effect on the eosinophiles tended to diminish after 4 to 5 weeks of hormone therapy. This was especially noticeable when a second course of treatment was administered.

Since it usually takes 3 days to bring the temperatures to normal in rheumatic fever when giving cortisone, it is wise to start with a large dose of the hormone (200 to 300 mgm.) the first day or two of treatment, and then reduce the dose so that by the 3rd or 4th day a maintenance dose of approximately 100 mgm. a day is arrived at. Since ACTH works more rapidly than cortisone, one can start with what one expects to be the maintenance dose and increase it in 3 to 4 days if necessary to adequately control signs and symptoms.

SUMMARY

Twenty-three cases of rheumatic fever were treated with ACTH or cortisone. Fever and arthritis were promptly relieved; the sedimentation rate was brought to normal in most cases in 3 weeks; the children gained on an average of 1½ pounds a week while on such therapy; gallop rhythm frequently disappeared; nodules disappeared in 3 cases in approximately 3 weeks; choreiform movements ceased in 3 weeks in 4 cases; there was usually an appreciable rise in hæmoglobin and hæmatocrit during treatment.

There was no significant change in heart murmurs. Changes in heart size were not significantly different from those seen in children treated with bed rest and salicylates. The sedimentation rate frequently rose after hormone therapy was stopped, but usually the rise was transient. In other cases the hormone had to be restarted for a further interval.

Patients with chronic heart failure were not improved by such therapy and in one case the failure became more marked during treatment. Two cases of acute heart failure in the first attack of rheumatic fever, were quickly and dramatically improved when hormone therapy was begun.

The authors wish to thank Miss McCallum, Miss Nesbitt, Miss Scott, Mrs. Robinson and Mrs. Thompson for their help in caring for the patients treated in this study, and for their assistance in recording the day-to-day data.

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RÉSUMÉ

Les auteurs rapportent vingt-trois cas de rhumatisme inflammatoire qui furent traités soit avec l'ACTH soit avec la cortisone. On injecta la cortisone à une dose quotidienne totale de 100 à 200 mgms en deux fois dans les 24 heures, et l'ACTH de 40 à 80 mgms par jour suivant le poids de l'enfant. La plupart des petits malades furent traités de 4 à 6 semaines, avec les résultats suivants qui font le sujet de ce rapport.

La fièvre disparut en moyenne en 2 jours avec la cortisone, et en 24 heures avec l'ACTH. La fluxion articulaire s'amenda en une journée ou deux. Dans la plupart des cas, le taux de sédimentation revint à la normale en 3 semaines, et les enfants dirent en moyenne des gains de 1½ livre par semaine tant que dura le traitement. Le bruit de galop cessa fréquemment de se faire entendre. Dans 3 cas les nodules disparurent dans environ 3 semaines, ainsi que les mouvements choréiformes; on nota une hausse habituelle de l'hémoglobine et des hématocrites. Pour ce qui est du cœur, il ne se produisit pas, ni dans les souffles ni dans l'augmentation de volume de l'organe, des changements importants différents de ceux que l'on observe chez les enfants dont le traitement consiste dans le repos au lit et la médication salicylée.

Le traitement à l'ACTH et à la cortisone n'améliore pas les petits malades en état d'insuffisance cardiaque chronique, dans un cas même il le rendit pire. Cependant deux sujets en état d'insuffisance cardiaque aiguë et qui en étaient à leur première crise de rhumatisme inflammatoire s'améliorèrent dès les premiers traitements d'une façon remarquablement rapide, voire dramatique.

THE PREVENTION OF RECURRING MANIC-DEPRESSIVE ILLNESSES*

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IT has long been generally believed that most people who have a manic-depressive illness will continue to have repeated attacks. That this is an error has been demonstrated by Fuller[1] and Pollock,[2] who found that less than half such patients had more than one attack. However, of all the mental disorders of a psychotic type, manic-depressive psychosis has a higher recurrence rate than any other.

* Presented to the London Academy of Medicine, September 28, 1950, and to the Medical Staff of the Veterans' Administration Hospital at Lyons, N.J., April 17, 1950.

Also, it has long been the hope of psychiatrists, that some way might be found to prevent such dreaded recurrences, because of the suffering they cause the patient and his family, the danger of suicide, the hazard of antisocial behaviour, or the risk to his estate through reckless extravagances, and the interference with his work and his career.

Although various efforts have been made to abort such recurring attacks in their prodromal phases, there has been no real success with such efforts, and until the advent of the newer physical therapies, such attacks usually ran their characteristic course, relatively uninfluenced by the therapies then available. With the use of the newer physical therapies, particularly electro-convulsive therapy and leucotomy, not only has the actual therapy of such mental illnesses been greatly improved, but it now appears that the hope of preventing recurrences is likely to be realized in at least a fair proportion of affected persons.

Our work at the Ontario Hospital, London, has led us to the conclusion that at least four methods have value in preventing recurrences.

1. The first method consists in the immediate administration of electro-convulsive therapy (hereafter to be designated as E.C.T.), at the first sign of an impending recurrence. The E.C.T. should be applied vigorously, two or three convulsions a day for several days, if necessary. In our experience E.C.T. is equally valuable for manic or depressive attacks, although manic attacks may require more vigorous therapy. Our first experience with E.C.T. to prevent regularly recurring manic attacks was reported by J. J. Geoghegan.[3] In this case, an impending manic attack was aborted by this method in a woman patient who had had recurring manic attacks for many years. Whereas previously she had been in severe manic attacks for several months each year, she has had only a very few days of mental illness during the past 5 years, as each successive attack has been successfully aborted by early and intensive application of E.C.T. In recent years we have been advising physicians and relatives of manic-depressive patients to be on the alert for beginning symptoms, and have been requesting them to place such patients under immediate psychiatric treatment, usually on an out-patient basis. A few such patients come to us of their own volition when they feel another attack is impending, requesting one or two E.C.T.'s. Although E.C.T. has its unpleasant aspects, nevertheless the intelligent patient much prefers one or two such treatments on an out-patient basis, to weeks or months of hospitalization which in all probability would include a course of E.C.T.

2. The second method is a modification of the use of E.C.T., which is called prophylactic E.C.T., and was devised at this hospital and reported on by Geoghegan[4] and Geoghegan and Stevenson.[5] Briefly, it consists of the administration of a single E.C.T. once a month after recovery from a manic-depressive illness. It is given to patients who have had two or more attacks in a five-year period, and is given monthly in order to dissipate accumulating tensions before they can appear as another clinical attack. Such prophylactic E.C.T.'s should be given over the ensuing five-year period, although at bi-monthly or tri-monthly intervals during the 4th, 5th and subsequent years. We are satisfied that this method can keep a patient with recurring manic-depressive tendencies free of actual recurrences and in good mental health. It has the advantage over the first method described, inasmuch as the patient does not have to be supervised closely and is in relatively little danger of having a recurrence. It has the disadvantage of requiring co-operation with a long series of monthly E.C.T.'s which can become rather tiresome. We have found it difficult to persuade patients to persevere in this program but those who do continue with it appreciate its insurance against future attacks. Our five-year study in the use of prophylactic E.C.T. is being published elsewhere (Stevenson and Geoghegan[6]). That this method has some measure of acceptance elsewhere is indicated in articles by Bennett[7] and Kalinowsky[8] and various personal communications.

3. The operative procedure of leucotomy (prefrontal lobotomy) is also of value in breaking the recurring tendency in some patients. In our leucotomy program we selected certain manic-depressive patients who had failed to make a good recovery on E.C.T., or who had failed to hold their recovery by showing rapid relapses, or who were having frequent admissions to hospital because of failure to co-operate with the prophylactic E.C.T. program. We had only the hope that this operation might be helpful, all other therapeutic means having proved unsuccessful, and we were agreeably surprised to find

that several such patients completed their recovery and have remained well since the operation. As the time interval since the operation is not much more than two years, we cannot be sure that a permanent cessation of attacks has resulted, but at least none of this group have as yet shown a recurrence. The following are a few illustrative cases.

CASE 1

A.L., 49, married, female. Recurring manic attacks between 1942 and 1947. Recovered from each attack but declined prophylactic E.C.T. Lobotomy August 26, 1948. Left hospital October 24, 1948. Recovered. No recurrence since operation.

CASE 2

M.M., 54, single, female. Admitted May 15, 1946. Extremely acute manic attacks with violence. Frequent improvements followed by relapses. Lobotomy August 5, 1948. Left hospital September 29, 1948. Has continued in excellent health. No recurrence since operation.

CASE 3

T.R., 40, married, female. Admitted October 28, 1946. Recurring uncontrolled excitements until lobotomy November 18, 1948. Left hospital March 13, 1949. No recurrence of symptoms. Excellent mental health. No recurrence since operation.

CASE 4

N.S., 58, single, female. Admitted January 11, 1943. Repeated depressions with improvement, always followed by relapse. Lobotomy (unilateral) July 22, 1948. Left hospital November 3, 1948. Excellent mental health with no recurrences since operation.

CASE 5

M.D., 31, single, female. Admitted December 16, 1942. Repeated depressions and suicidal thoughts. Lobotomy April 29, 1948. Left hospital June 23, 1948. Steadily and happily employed. No recurrence of symptoms since operation.

4. The fourth method of value in preventing recurrences involves a successful solution of the patient's environmental difficulties, by his own efforts, the efforts of his family, and particularly by an efficient After-care Department. It is our belief that chronic frustration is an important factor in many manic-depressive recurrences, hence the need of an adequate social psychotherapy. The only two failures in our prophylactic E.C.T. group presented insoluble social frustrations. The satisfactory social and personal rehabilitation of recovered manic-depressive patients is one of the important tasks of our After-care Department. It is too early to present any statistical evidence as to the value of an After-care Department in a mental hospital, but we have reason to believe it has genuine value, and federal grants have been made available to us to develop this potentially important feature of the more adequate rehabitation of recovered mentally ill patients.

SUMMARY AND CONCLUSIONS

1. Somewhat less than half the patients afflicted by manic-depressive psychosis have recurrences.

2. Pessimism with reference to the inevitability of recurring manic-depressive illnesses, even in persons who have had repeated attacks, is no longer warranted.

3. Many recurrences in the manic-depressive group may be prevented by the use of one or more of the following techniques: (*a*) Intensive early application of E.C.T. when a recurrent attack is impending. (*b*) Prophylactic E.C.T. to recovered patients, consisting of a single E.C.T. each month, with gradual increase in the interval between treatments. (*c*) Leucotomy in selected cases. (*d*) Adequate after-care by a trained after-care department and a social psychotherapy to assist in solving the patient's frustrations.

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SURGICAL TREATMENT OF CHRONIC ULCERATIVE COLITIS*

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THERE has been a change in the therapy of idiopathic chronic ulcerative colitis in recent years but the primary treatment for this disease still consists of a carefully regulated medical regimen. It has been variously estimated that 25 to 30% of patients fail to respond to this type of management. The hope for relief in this latter group lies in surgical intervention and it is with these problem cases that we are primarily concerned in this discussion. Acceptance of radical surgical measures as a mode of therapy in these difficult cases is not easy. Patients and many phy-

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sicians are not prepared to accept permanent ileostomy as the price of a successful result. The reports of Cave, Cattell, Dennis and others show that the late results of surgical therapy are superior to medical management in the intractable cases in this disease entity.

The etiology of ulcerative colitis is unknown. It is now considered a distinct clinical entity and most observers feel that it is not related to either amœbic or bacillary dysentery. Bacteriological, allergic, enzymatic, and psychological factors have been presented as important etiological agents by various observers.

The patients form two groups: (*a*) those with diffuse involvement of the whole colon, the so-called Type 1 of Bargen, 95% fall into this group; and (*b*) with an isolated segment of bowel involved the terminal portion of the colon being free of disease, the so-called Type 2 of Bargen. Normal rectal mucosa is pinkish in colour with a smooth glistening surface through which a network of venules may be seen. In the earliest cases of ulcerative colitis the mucosa appears diffusely red and œdematous but is not ulcerated. The thickened mucosa narrows the bowel lumen and obliterates the venules. The swollen mucosa may present multiple hæmorrhages arising from ruptured capillaries. This picture may come and go as the symptoms present or relapse. The x-ray picture at this stage shows hyper-irritability of the bowel. The haustral markings may be smoothed out but there may be little or no abnormality in the appearance of the colon. Symptoms usually consist of gradual increase in the number of stools and in the fluidity of stools. There may or may not be blood in the stools and fever is usually absent. Amœba must be carefully excluded. Chronic ulcerative colitis, then, in its mildest form is a non-febrile, non-toxic condition characterized by an inflammatory reaction in the colon.

The active inflammatory reaction may subside or it may progress either rapidly or slowly to ulceration of the mucosa. In later stages little or no normal mucosa may be seen. Ulceration, of course, denotes a break-down of tissue resistance followed by an opening up of lymph channels to secondary invasion by a host of organisms which are always present in the bowel. With this invasion of secondary organisms the clinical features change. The bowel symptoms increase and systemic reactions are added. Fever, septic in character, malaise, increase in leucocyte count, increase in sedimentation rate, purulent rectal discharge, anorexia, vomiting, etc., indicate marked toxicity. At any stage regression may occur, healing with replacement fibrosis taking place. Each attack adds to the scarring of the bowel so that it eventually becomes rigid and tube-like; the so-called "pipestem" colon of long-standing chronic ulcerative colitis. If the progress of the acute inflammation is fast, we see the so-called acute fulminating type of lesion with severe diarrhœa, and loss of blood, inanition, sepsis and frequently death.

The diagnosis of this disease process rests on proctoscopic examination, the character of the stool and the x-ray examination. Barium enema examination early as we have stated, may appear very nearly normal and at a little later stage there may be a feathery deformity of the mucosal pattern. Still later the tube-like character of the bowel is evident, the colon and the mesentery markedly shortened and the lumen narrowed.

There is no specific or reliable medical cure for ulcerative colitis. Improvement in nutrition, control of diarrhœa, treatment of the anæmia and hypo-proteinæmia help to promote remission of active disease. It is more accurate to speak of medical control of the disease rather than cure.

What are the indications for surgical intervention? It seems to me that the indications for surgery in this disease are very similar to those in duodenal ulcer, that is, for the complications of the disease process. In ulcer they are usually listed as perforation, obstruction, hæmorrhage and intractability. In chronic ulcerative colitis I feel we should discuss them in the reverse order. Let us consider intractability or chronic invalidism. Here again as in ulcer the indications are not clear-cut and close co-operation between internist and surgeon is of great importance. When a patient fails to show satisfactory improvement with adequate medical management and is not able to return to work or lead an essentially normal life, something must be done to rehabilitate him. A large percentage of these patients can be handled surgically, returning the patient to his occupation and normal place in society. For the most part, however, no patient should be

subjected to surgery for so-called intractable ulcerative colitis who has not known the ravages of the disease for a prolonged period. If suffering has been in keeping with that usually experienced, the acceptance of permanent ileostomy is gratifying in most cases.

Hæmorrhage, that is massive hæmorrhage, is fortunately rare in ulcerative colitis but case reports indicate that ileostomy in itself may not be the answer in this complication but an emergency colectomy may be indicated to control blood loss.

In long-standing cases marked fibrosis and contraction of the diseased colon may lead to obstruction. Ileostomy with or without partial colectomy, is the primary treatment in these cases.

Free perforation occurs frequently in the fulminating cases and this complication carries a very high mortality. If on the other hand perforation occurs slowly, walling off of the area may occur and a localized abscess rather than a spreading peritonitis results. Internal fistulæ to urinary bladder or another loop of bowel may follow. If perforation occurs below the peritoneum, perirectal or ischio-rectal abscess may occur with sinus formation and very often incontinence. Diversion of the fæcal stream is certainly indicated in this group. Approximately 6% of cases present complications in the anal region. Four per cent of cases exhibit perforation above the peritoneum commonly in the transverse and descending colon.

In addition to these indications the incidence of malignancy in colons affected by chronic ulcerative colitis is noticeably higher than normal. Sloan, Bargen, *et al.* have recently shown that there is a 19% incidence of polyps in this disease. These polyps had for years been regarded as pseudopolyps or inflammatory tags of mucosa following an extensive ulcerative process. This study has demonstrated that 20% of the cases in which polyps develop exhibit true adenomatous changes. These adenomatous changes are usually recognized grossly by the exuberant vascular appearance of the polyp in distinction to the pale fibrous appearance of the pseudopolyp. Bargen has emphasized the fact that malignant disease occurs almost exclusively in this group in which true adenomatous polyps develop. In this recent study of 2,000 cases the incidence of malignancy was 5%.

Colcock feels that ileostomy may have a place early in acute fulminating cases and Dennis has reported successful management of two of these cases of acute fulminating type of vagotomy. We should perhaps keep an open mind with reference to the use of surgery in this desperate situation.

Localized lesions in the colon respond well to surgical excision but they are very rare. Operation should be carried out early to prevent spread of the disease.

When surgery has been elected, preoperative preparation includes correcting nutritional deficiencies to the extent that this is possible by diet in the presence of the disease. Much is written about high protein diet in the preparation of these people, but in our limited experience diarrhœa increases in the attempt to force-feed them. We have had to depend on blood transfusions, plasma, intravenous amino acids, and so on, to attempt to raise the serum protein level. Vitamine C, K, and B complex are indicated and a course of antibiotics to eliminate secondary invaders and control infection. These patients should be in fluid balance and blood chemistry studies should be within normal limits before operation.

Ileostomy after a hectic early postoperative course may be followed by a dramatic response in the patient's condition. This diversion of the fæcal stream may result in the healing of the inflammatory process and in a very few cases gastro-intestinal continuity may be re-established at a later date. This as a rule should not be anticipated and when it is possible Thorlakson is of the opinion that the original ileostomy was ill-advised.

Ileostomy is usually carried out through a left paramedian incision in the lower abdomen. The ileum is transected at least 30 cm. from the ileo-cæcal valve in accordance with the findings of Cattell and McCready *et al.* These observers found a very high incidence of ileal involvement averaging 20 cm. in one series. We now ask the pathologist to examine a piece of bowel just distal to the point of transection to confirm its being free of disease. The proximal ileum is brought out through a stab wound about 5 to 6 cm. below the umbilicus and 2 to 3 cm. to the right. This seems to be the most comfortable place for the patient to fit the Koenig-Rutzen bag. We do not replace the distal ileal loop into the peritoneal cavity

but bring it out high in the left paramedian incision so that it will be in the line of incision for later colectomy. In 1946 although I performed only two ileostomies for ulcerative colitis my operative experience in revising these ileostomies was extensive. In the first case revision of the ileostomy had to be carried out twice because of recurrent prolapse with obstruction to the ileal flow on both occasions. The second ileostomy had to be revised twice for obstruction, once at the peritoneal level and once at the skin level. However, our results have improved since then and in our last few cases we have adopted a simple procedure recommended by Hart. The mesentery of the terminal ileum is spread out over the posterior peritoneum in the right iliac fossa as an open fan with the terminal ileum coming across the pelvis from right to left and extending up and out to the ileostomy opening. The mesentery is then anchored to the posterior peritoneum and right lateral peritoneum with mattress sutures of silk taking care to avoid injury to blood vessels. With this anchoring, prolapse is impossible and our results have been entirely satisfactory.

For some years Lahey and his co-workers have combined first stage colectomy, that is to the midtransverse colon, with ileostomy in patients who are in the chronic cicatricial stage of the disease. Gavin Miller and his associates in Montreal have extended this to the point where they advocate ileostomy plus right and left colon resection as a primary procedure in all patients in whom surgery is indicated in this disease. Their preliminary reports are encouraging and justify their adoption of this radical procedure.

A complete transverse incision is useful in the performance of colectomy. The rectal stump is removed if indicated with an abdomino-perineal procedure and in a very limited experience we have found the two team method most useful.

Resorption of fluid from the fæcal stream is the function of the colon. After ileostomy an adjustment must be made to the loss of this resorptive surface. For the first few days fluid and electrolyte loss is great through the ileostomy and great care is necessary to maintain fluid and electrolyte balance. Blood chemistry studies and urinary output will indicate the replacement necessary. In one of our recent cases potassium deficiency was a very marked feature and careful replacement was essential over a period of weeks. The small bowel eventually takes over the function of resorption of fluid for these people.

Excoriation of skin is an unpleasant and troublesome complication of ileostomy. Agreement is universal that prevention is far superior to our attempts to alleviate the situation after it has developed. A variety of methods have been advocated. Kaogel ointment has replaced all other local applications in our routine. The development of a well-fitting ileostomy bag that permits an airtight connection between the bag and the abdominal wall has been one of the outstanding factors in the restoration of these patients to normal social and economic activity.

Clarence Dennis has recently advocated the use of vagotomy in the treatment of this disease. The rationale of this procedure is:

1. Relief of enteric spasm.
2. Reduction of motility of the entire intestine.
3. The apparent blocking of the rectosigmoid response to emotional stress.
4. The observation of Lillehei and his associates that fatal diarrhœa seen in dogs after mesenteric ganglionectomy does not occur if the vagi are divided. Dennis classes vagotomy as a conservative measure and reserves the operation for young people who still have flexible colons, where the disease is of relatively short duration with little radiological evidence of fibrosis. It is too early to evaluate the procedure but Dennis' results would indicate that it was a justifiable experiment and the initial response in some of his early cases was astounding. One cannot but feel that very careful postoperative follow-up on these patients is essential in view of the possibility of malignancy developing.

A 31 year old married woman who had been suffering from chronic ulcerative colitis for 3½ years was admitted to our hospital in January, 1949. She had been in hospital on 4 occasions varying from three to seven weeks during the course of her illness. Her hæmoglobin was 30% on admission and sigmoidoscopically she had a typical picture of chronic ulcerative colitis. Stool studies were negative for amœba and the x-ray picture was typical of ulcerative colitis but the bowel was distensible. In other words, there was still some flexibility of the colon. She was having ten to twelve stools a day and was thoroughly miserable.

She refused ileostomy and for this reason after adequate preparation on January 13, 1949, she had a transthoracic vagotomy carried out at the University of Alberta Hospital. Her immediate postoperative response was dramatic. Her stools dropped to two to

three a day immediately postoperatively and she has had no blood in her stool since the operation. On May 23, 1950, sixteen months after operation, she returned for assessment. She had gained 30 lb., her stools while soft, did not occur more than twice a day and she has days when she has none. She looks and feels well. Sigmoidoscopic examination revealed ulcerative colitis.

In view of the result in this patient, one of our conservative internists, Dr. Frank Elliott, asked us to see another 26 year old girl with a similar type of history and similar findings. It is only a short time since she was operated upon but her immediate response was just as satisfactory. We realize full well these two cases mean little except that these two people have been greatly improved and have been returned so far to normal health.

Our very small series of cases of chronic ulcerative colitis is not of any value statistically or in the evaluation of surgery in this disease but McKell recently questioned 84 patients with permanent ileostomy. 79 were pleased with the results and said they wished it had been done earlier. McKittrick and Moore point out that 70 to 90% of their patients consider the end result of ileostomy satisfactory. These reports and our own small experience make one feel that surgical management in intractable ulcerative colitis is productive of better health than prolonged conservative treatment. I am convinced that many patients who now plan their daily activities from toilet to toilet could be made much happier with a properly functioning ileostomy and a satisfactorily fitting Koenig-Rutzen bag.

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The more things a man is ashamed of, the more respectable he is.—George Bernard Shaw.

INTERNAL MAMMARY CORONARY ANASTOMOSIS IN THE SURGICAL TREATMENT OF CORONARY ARTERY INSUFFICIENCY*

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THIS paper constitutes a preliminary report of clinical cases which have undergone transplantation of the left internal mammary artery into the left ventricle as a treatment for coronary artery insufficiency. The theoretical and experimental basis on which this procedure is based will be briefly described.

EXPERIMENTAL DATA

Many attempts have been made experimentally to improve ventricular myocardial circulation such as, the application of fat or muscle grafts to the heart, the use of irritating foreign bodies such as talc, or asbestos fibres to produce adhesions between pericardium and myocardium. Fauteux[1] attempted to improve the myocardial circulation by cardiac vein ligation and Beck[2] by means of arterialization of the coronary sinus. One of us (A.V.) has attempted to obtain this improvement by the direct implantation of a living artery, namely the left internal mammary artery into the left ventricular myocardium. The artery is placed within a tunnel in the myocardium and in over 200 experiments the degree and frequency of new anastomoses have been experimentally proved by injection studies, radio-graphs, plastic casts and serial sections.[3, 4] Anastomosis occurred in 50 to 75% of these animals, depending on the technique of implantation used. No animal developed infarction or died following anterior descending branch ligation when a large anastomosis had developed. All these experiments have been controlled and the control group, without implantation of a living vessel into the heart muscle, showed a mortality of 90%, and in 10% a large infarction developed following the same ligation of the anterior descending branch.[5, 6] (It is recognized that thrombosis of the anterior descending branch of the left coronary artery is the most common cause of death in human coronary artery disease.)

The anastomoses which developed have been shown both by injection and by histological serial section to be true arterial branches. It has been reported by Glenn[7] that these branches only live for 8 weeks. Our observations have definitely shown that they last at least 58 weeks which is the longest observation made before the animal was sacrificed to confirm the persistence of these vessels.[8] The direction of blood flow through the implanted internal mammary artery was studied in order to determine whether or not blood was being brought to the ventricular myocardium through the internal mammary artery. Direct determination of blood flow in the internal mammary artery was difficult, so the indirect method was used. Animals which survived anterior descending branch ligation of the left coronary artery were subjected after 4 weeks to complete and sudden occlusion of the implanted internal mammary artery. If the internal mammary artery was maintaining the circulation of the left ventricle then, following its ligation, either death or infarction should result. This is exactly what happened. In three animals with an internal mammary implant that had survived anterior descending branch ligation, the internal mammary artery was ligated. One animal died within 24

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hours and displayed an œdematous cyanotic area of the anterior wall of the left ventricle. One survived for 3 days before dying from a large infarct in the same location. The 3rd animal survived, but examination of the sacrificed specimen revealed that multiple intercoronary anastomoses were present.

The experimental results just described were obtained by implanting the internal mammary artery into normal dogs' hearts. It was suggested that in human coronary insufficiency such a procedure would be of little value because of the presence of occluded coronary vessels and associated myocardial ischæmia. It was, therefore, decided to experimentally produce coronary artery insufficiency in the dog. This was done by wrapping the origin of the anterior descending branch of the left coronary artery with a sclerosing type of cellophane.[9] The cellophane surrounding the origin of the anterior descending branch of the left coronary artery set up a periarterial fibroplasia which resulted in a gradual contraction through scar tissue of the coronary artery with narrowing of its lumen. This caused a reduction in blood flow through the narrowed vessel, and resulted in ischæmia of the left ventricular muscle supplied by the anterior descending branch of the left coronary artery. The degree of myocardial ischæmia was evaluated by estimating the exercise tolerance of the animal on a motorized treadmill. Before the cellophane wrap was placed around the coronary artery the animals would run 9 to 12 minutes at 8½ miles per hour on the treadmill. With this amount of exercise the animals became tired, began to lag on the mill and would break alternatively from a gallop to a run. Eventually they would become anxious to escape the mill. When the exercise was discontinued these animals would pant, but would drink water and appear similar to any other normal dog after exercise. Five months after these animals had had a piece of cellophane wrapped around their anterior descending branch of the left coronary artery they would run for 1 6/10 minutes on the treadmill before becoming extremely anxious, begin to whine and salivate profusely. If the exercise was not terminated they would attempt to lie down or drag their feet on the revolving platform. When the mill was stopped they would drop where they stood and resist all coaxing to move for some minutes and would not drink water. Animals that had reached this stage were then subjected to a left internal mammary artery implant. Four months after the implant those animals which had definitely developed an internal mammary coronary anastomosis had a return of exercise tolerance to 7 minutes or more. This occurred in spite of a completely occluded anterior descending branch of the left coronary artery. When an internal mammary coronary anastomosis failed to develop there was no improvement in exercise tolerance in such animals after implantation.

Because of these experimental results it was thought that implantation of the internal mammary artery into the left ventricular myocardium might be of value in the treatment of human cases of coronary artery insufficiency.

HUMAN CASES OF CORONARY ARTERY INSUFFICIENCY TREATED BY INTERNAL MAMMARY ARTERY TRANSPLANT

Selection of cases.—The estimation of clinical results is always difficult and the results of any given surgical procedure may vary according to the severity of the disease process at the time of operation. There are certain well-known pathological facts concerning coronary artery sclerosis and thrombosis which greatly influence the selection of cases for internal mammary artery implant. Perhaps the most important is the fact that coronary artery sclerosis in general is confined to the first 3 or 4 cm. of the coronary artery. It has been stated that arteries beyond the first 3 or 4 cm. of the coronary vessels show lesser degrees of sclerosis and rarely is sclerosis seen after the 3rd or 4th order of branching. Sections have shown that in cases of severe coronary artery sclerosis the vessels lying within the heart muscle are generally free of arterial disease. Thus, an internal mammary artery placed in the ventricular muscle is placed in an area where the arteries are comparatively healthy. In this way fresh blood can be brought to the network of non-sclerosed arteries and arteries which exist beyond the points of coronary artery obstructions.

In cases of coronary artery thrombosis with myocardial infarct,[10] the picture is entirely different. In these cases, if the patient survives, an area of myocardial infarction the muscle undergoes degeneration and eventually heals by scar formation. In most cases there will be healthy muscle surrounding the area of the healed infarct. It is also stated that during the process of healing new blood vessels grow into the infarcted area; thus the history of a left coronary artery thrombosis with recovery does not constitute a contraindication to internal mammary artery implantation. The new living arteries can be placed in healthy muscle which is present at the edge of the healed infarct and, if necessary, into the intraventricular septum itself. With this in mind, it is clear that patients who have recovered from coronary artery thrombosis and infarction may be considered as candidates for an internal mammary artery transplant. Our last patient was known to have had two attacks of coronary artery thrombosis with a posterior wall infarct. At operation there was still what appeared to be good muscle posteriorly. There was, however, evidence of scarring on the posterior surface towards the apex which extended for a half inch or more to the anterior surface of the left ventricle. The internal mammary artery was placed into healthy muscle which was present just proximal to this scarred area. We have been reluctant to operate upon patients who are able to carry on their normal daily activities. We have to date only operated upon those patients who are unable to carry on because of the severity of their anginal pain. Patients with an enlarged left ventricle and myocardial decompensation have been considered poor risks and have not been accepted. Every attempt has been made to exclude other sources

of anginal pain and to make certain that the pain from which the patient is suffering is due to coronary insufficiency. Where there is a doubt and particularly when other organic disease such as cholelithiasis exists, the associated disease has been treated first.*

Preoperative investigation.—Each patient has undergone extensive investigation in order to establish a diagnosis of coronary insufficiency and to determine its extent. A careful clinical history has been supplemented by detailed electrocardiographic studies made at rest and after exercise. In order to correlate the anginal pain with myocardial ischæmia electrocardiograms have been taken after exercise at the height of the anginal pain. A record was made on each patient as to the extent of his exercise tolerance prior to operation as indicated by the number of stairs he could climb in a given time before experiencing the onset of anginal pain.

In each patient a careful survey has been made to exclude sources of pain which were not cardiac in origin. Complete radiographic studies have been made of the œsophagus, stomach, duodenum and gallbladder, as well as of the thorax and lungs. In each case a Mosenthal test, non-protein nitrogen, basal metabolic rate, blood cholesterol, blood sugar and hæmograms were also recorded.

CASE 1

Mr. J.P., (referred by Dr. L. I. Frohlich of Montreal) age 53, occupation, tailor. Admitted to the Royal Victoria Hospital, April 24, 1950. On admission the patient's chief complaint was that of substernal pain which radiated to the left jaw and down the left arm to the wrist. Occasionally the pain radiated to the back in the interscapular region. The pain was brought on by exertion and emotion. It was an aching, pressure type of pain, and was not very sharp. It was first noticed 14 years ago, gradually becoming more severe. The patient had been unable to work for three years prior to admission and had suffered pain day and night. Some, but not complete, relief was obtained by nitroglycerin. Exercise tolerance was limited to one city bock; walked very slowly before onset of anginal pain occurred. There was a history of 40 pounds loss of weight in the past 10 years. There was also a history of intolerance to fatty foods with eructations of gas and inability to eat a large meal.

General physical examination revealed a well nourished white male of good colour. Temperature was 98, pulse 80, respiration 20, blood pressure 120/90. There was a right, direct inguinal hernia. There were bilateral varicose veins.

Exercise tolerance test.—The patient developed pain after going up 22 steps in a 10 minute period. The electro-cardiograms, taken before and after exercise, are shown in Figs. 1 and 2 respectively. Blood and spinal fluid serology were negative. The lungs, œsophagus, stomach, duodenum and gallbladder were radiologically normal. Blood analysis for sugar and cholesterol were found to be within normal limits. There was no increase in white blood count or the sedimentation rate.

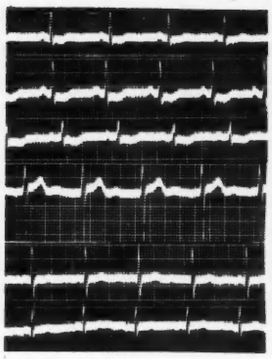

Fig. 1

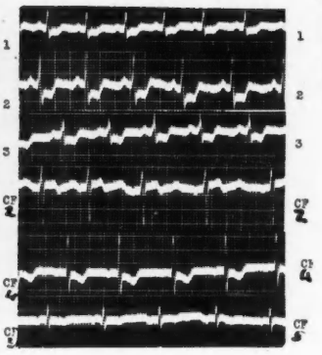

Fig. 2

Fig. 1.—Electrocardiogram taken after 3 hours' rest in bed. A slurring of QRS complexes. Depression of S-T interval in leads 1, 2 and 3. Lead CF_2 normal. CF_4 shows a low T-wave as does CF_5. Myocardial changes with coronary insufficiency. Rate 60 per minute. **Fig. 2.**—Electrocardiogram taken at height of pain after 22 steps climbed in 10 minutes. The standard leads show a marked depression of the S-T intervals after exercise. A marked coronary insufficiency. The CF leads show a diphasic T-wave. CF_4 a negative T and a shallow negative T in CF_5. Evidence of marked coronary insufficiency. Rate 90.

Operation.—On April 28, 1950, implantation of the left internal mammary artery into the left ventricular myocardium was carried out. Prior to the commencement of the anæsthetic, the patient experienced severe substernal pain which was not relieved by two tablets of nitroglycerin. An electrocardiogram taken at this time did not show any changes indicative of coronary thrombosis. The blood pressure was unaltered so it was decided to proceed with the operation. The left thorax was entered through the 5th intercostal space by an anterolateral approach. The 4th and 5th ribs were resected subperiostally 10 cm. lateral to the sternum, including part of their cartilages. Approximately 1.5 cm. of cartilage was left in position. Procaine 1% was injected into the pericardium and was also given intravenously as a continuous drip. At this time the blood pressure, which had slowly been dropping, was recorded at 70/50. Patient was placed in the Trendelenberg position and the blood pressure returned to 90/70. The internal mammary artery was freed from the chest wall between the 4th and 6th intercostal spaces. The intercostal arteries 4th and 5th were doubly ligated with 000 catgut. The internal mammary artery was tied with cotton and severed between ligatures. The pericardium was opened. The left ventricular muscle was firm and was covered by a layer of fat. A traction

* The final decision in the selection of each patient for operation was made jointly with Dr. G. R. Brow, Director of the Department of Medicine.

suture was placed in the apex of the heart. The 6th intercostal artery was cut and bled freely and was pulled with the internal mammary artery into a tunnel in the myocardium. The internal mammary artery was held in position by a traction suture which was tied about it. The entire procedure of implantation took about 3 minutes. There was little evidence of ventricular irritability or arrhythmia. The blood pressure at the time of implantation was 70/60 which quickly returned to 110/80 after the thorax was closed. The pulse rate throughout the operation was comparatively slow at about 100 per minute. The left thorax was drained. The total blood loss during operation was 260 c.c., as measured by the gravimetric method.

Postoperative course.—For the first 12 hours after operation the blood pressure remained steady at 110/72 with a pulse rate of 108 per minute. The patient was conscious and appeared clinically quite well. Gradually the blood pressure sank until 24 hours after operation it was 86/62. The pulse rate, however, had dropped to 96 per minute and the patient's general condition was excellent. He was removed from the oxygen tent and given a liquid diet. An electrocardiogram taken approximately 24 hours after operation showed anterior myocardial changes and coronary insufficiency, but the rhythm was regular, and the a-v conduction time was normal. In spite of the low blood pressure the patient's general condition remained excellent throughout the succeeding two days. At 5.54 p.m., April 30 he attempted to use the bed pan. This was followed by a rapid drop in blood pressure and elevation of pulse rate to 160 per minute. On May 1, at 12.30 a.m., chest pain developed and there was an increase of the respiration rate to 22. At 1.25 a.m. the chest pain became more severe and the condition of the patient rapidly deteriorated. He expired at 1.45 p.m.

The interval between completion of operation and death was a little over 2½ days.

PATHOLOGICAL FINDINGS

The three main coronary arteries were pipestem in character for their first 3 or 4 cm. All the coronary arteries showed multiple areas of marked stenosis due to arteriosclerotic plaquing. The right coronary artery was completely blocked by an old, firm, greyish thrombosis. The left anterior descending branch was completely occluded for a distance of 1 cm. by a recent, soft, dark-brown thrombosis. The anterior wall of the left ventricle and intraventricular septum showed evidence of recent infarction.

The internal mammary artery which had been implanted was patent throughout. There was no hæmatoma at the site of implant (see Fig. 3).

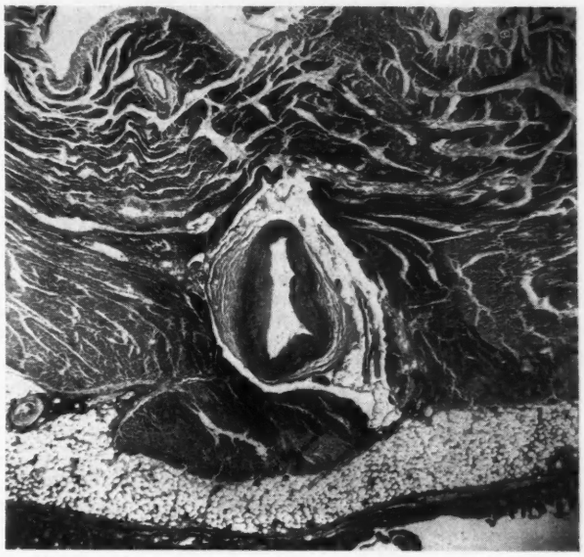

Fig. 3.—Shows the internal mammary artery lying in the human myocardium 62 hours after implantation.

Death was due to a recent thrombosis of the anterior descending branch of the left coronary artery with ventricular infarction. There was an associated atelectasis of the left lower lobe.

CASE 2

Mr. D.M., age 54. Admitted to the Royal Victoria Hospital, October 22, 1950. Discharged December 5, 1950. This patient, 7 months prior to admission, developed severe precordial pains which radiated down the left arm. Pain was initiated by exercise and was associated with shortness of breath. Pain frequently followed the ingestion of solid food. This was so pronounced that for a few months prior to admission patient lived on a liquid diet. His exercise tolerance was limited to about one city block. There was a bad family history. His father and two paternal uncles died of heart disease. One brother had had coronary artery thrombosis.

Physical examination revealed a well nourished slightly obese adult male with sallow complexion and a somewhat myxœdematous appearance to his face. The abdomen was pendulous due to excessive fat. Pulse 68 to 74, temperature 98, respiration 18, blood pressure ranged between 140/100 to 106/60.

Exercise tolerance.—Fifty-six steps with 7" elevation were climbed in 2½ minutes before pain in the precordium and left arm occurred. A control electrocardiogram taken at rest is shown in Fig. 4, and an electrocardiogram exercise taken at the height of pain is shown in Fig. 5.

Blood serology and chemistry for sugar and non-protein nitrogen were normal. Radiographic studies of the lungs, œsophagus, stomach, duodenum and gall-bladder were normal. There was no elevation of the white count or sedimentation rate. The Mosenthal test showed excellent concentration and output. The basal metabolic rate was normal.

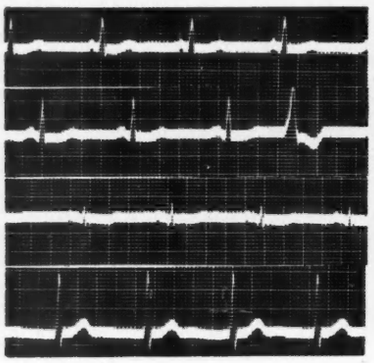

Fig. 4

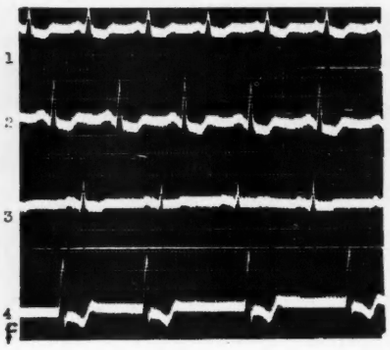

Fig. 5

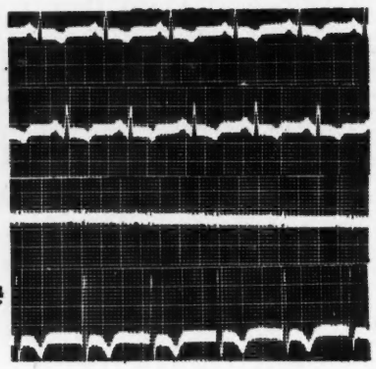

Fig. 6

Fig. 4.—Sinus rhythm. Normal auriculo-ventricular conduction time. QRS complexes slurred. Right ventricular extra systoles in lead II. Low voltage T-waves in all leads. Diphasic P-waves in lead III. Ventricular rate 60 per minute. Lead 4F adds nothing more. **Fig. 5.**—Regular rhythm. Normal auriculo-ventricular conduction time. QRS complexes slurred. R-T intervals depressed in leads I and II. Diphasic T-waves in lead III. Ventricular rate 90 per minute. Lead 4F shows a depressed S-T and negative T-waves. Coronary insufficiency. **Fig. 6.**—Regular rhythm. Normal auriculo-ventricular conduction time. Negative T-waves in leads I and II. QRS complexes slurred. Ventricular rate 90 per minute. Lead 4F shows an acutely inverted T-wave. The picture is that of recent anterior infarction.

Operation.—In this patient and in the subsequent case, the blood pressure was maintained throughout the operation by means of a continuous intravenous drip of neosynephrin. The blood pressure was 130/60 at the commencement of the anæsthetic and was never permitted to drop below 120/70 throughout the entire operation which lasted approximately 2½ hours. It was necessary to increase the rate of flow of neosynephrin at various times during the operation in order to prevent the blood pressure from dropping. The technique followed was approximately the same as that described in the first case, except for the approach which was made through the 5th intercostal space with severance of the 4th and 5th ribs just distal to the costal cartilages. The heart at the time of implant showed little disturbance. The blood pressure was maintained throughout. There was no fall in blood pressure during the operation such as occurred in the first patient. The anæsthetic used was cyclopropane and ether.

Postoperative convalescence.—The immediate postoperative blood pressure was well maintained and varied from 142/106 to 100/70. At the time of discharge, the blood pressure varied from 140/80 to 100/74. The pulse rate 48 hours after operation reached 120 per minute. This slowly diminished, and at time of discharge was between 80 and 90. The postoperative convalescence was essentially uneventful except for the complication of a paralytic ileus which was easily controlled by Wangensteen drainage. There was also a diffuse pleuritis of the left thorax which gradually improved.

The patient was allowed to sit on the side of the bed on the 10th postoperative day, and was permitted to sit in a chair on the 23rd day. He was discharged to his home five weeks after operation. At the time of his discharge he was able to eat solid food and walk slowly. There was no anginal pain after eating or during walking. An electrocardiogram taken the day before discharge is shown in Fig. 6. Recent communication from this patient states that he is completely free of pain and working.

CASE 3

Mr. E.S., age 49. Admitted to the Royal Victoria Hospital, November 9, 1950. Discharged December 18, 1950. This patient, in December, 1947, developed uræmia and was extremely ill. In 1948 after playing 27 holes of golf, he experienced severe pain in his left arm which radiated to the chest. A diagnosis of left posterior branch coronary occlusion was made. Since that time he has suffered from anginal pain which radiated up his left arm to the precordial region and sternum. Occasionally, the pain went into the throat and jaw. The anginal-like pain was initiated by any change of pace. It was particularly severe on getting out of bed in the morning or on walking rapidly. It was relieved by nitroglycerin. Sometimes it was so severe that patient was forced to take demerol.

Temperature was 97.2, pulse rate varied from 112 to 90, respirations 18, blood pressure varied from 174/120 to 120/90. Physical examination was essentially normal.

Exercise tolerance.—Ninety steps were taken in 1½ minutes without increasing the pain. The pain, however, was present before starting the exercise. Control and exercise electrocardiograms are shown in Figs. 7 and 8. Blood chemistry for serum cholesterol, sugar and non-protein nitrogen were within normal limits. The basal metabolic rate, hæmogram and Mosenthal test were normal. Radiographic examination of the lungs, œsophagus, stomach, duodenum and gallbladder were normal.

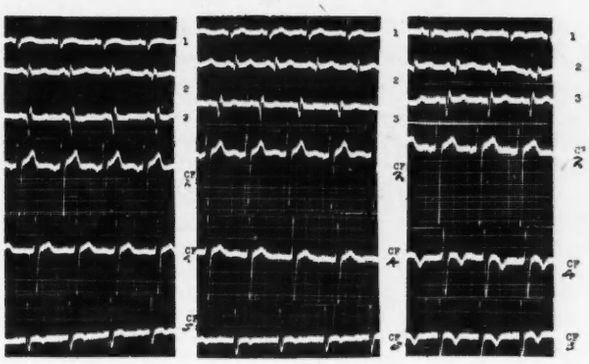

Fig. 7 Fig. 8 Fig. 9

Fig. 7.—Regular rhythm. Normal auriculo-ventricular conduction time. Left-sided preponderance. QRS complexes slurred. Q-wave in leads II and III. T-wave flattened in all standard leads. Ventricular rate 90 per minute. Leads CF_2 and 4 add nothing. CF_5 shows negative T-waves. Myocardial changes. Coronary insufficiency. **Fig. 8.**—Regular rhythm. Normal auriculo-ventricular conduction time. QRS complexes slurred; Q-waves in leads II and III. T-waves of greater voltage. Ventricular rate 90 per minute. Leads CF_2, CF_4 and CF_5 are unchanged. **Fig. 9.**—Regular rhythm. Normal A-V conduction time. Left-sided preponderance. QRS complexes slurred. Q waves in leads 2, 3. Negative T-waves with elevated S-T in lead 1. R-T elevated in lead 3. Ventricular rate 90/min. Leads CF_4 and CF_5 show an acutely negative T-wave. Anterior myocardial infarction.

Operation.—On November 20, 1950, under cyclopropane and ether, an internal mammary artery implant was carried out. The blood pressure at the commencement of the anæsthetic was 130/80, and was quite difficult to maintain before and during operation. Large amounts of neosynephrin were given in order to keep the blood pressure at approximately 130/80. A total of 4 c.c. of 1% neosynephrin was used in this case from the time the anæsthetic was commenced to the conclusion of the anæsthetic. The pulse rate at the beginning of the operation was 110 per minute. It climbed to 140 and 150 per minute and then settled down to approximately 130 per minute for the duration of the procedure. The operative technique followed was essentially the same as in the second case, except for the fact that the 5th rib and costal cartilage were removed, which facilitated exposure. The implantation of the left internal mammary artery into the myocardium of the left ventricle caused little cardiac disturbance. It should be noted here that in this case and in the second case the 6th intercostal arteries were not bleeding at the time of implant.

Postoperative convalescence.—The pulse rate reached 130 at the end of 48 hours. This slowly settled down to vary between 80 and 100 where it was at the time of his discharge. The immediate postoperative blood pressure varied between 142/100 to 100/72. This patient developed a paralytic ileus and a patch of pneumonia in the right lower lobe. Paralytic ileus was treated by means of gastric and duodenal decompression and the pneumonia with streptomycin and aureomycin. The patient was given 3 grains of quinidine before and after operation. He was permitted out of bed at the end of three weeks and returned to his home four weeks after the operation. At the time of his discharge he showed marked improvement of his anginal pectoris. There remained only a slight pain in the left wrist upon getting out of bed in the morning. This did not require nitroglycerin or demerol. The discharge electrocardiogram is shown in Fig. 9.

DISCUSSION

In our experimental work it has been shown that when the internal mammary artery is placed in the ventricular myocardium it forms new arterial branches which anastomose with the left coronary circulation. When this occurs, the heart is protected against death by infarction following the occlusion of the anterior descending branch of the left coronary artery. It has also been shown that those animals which have survived a ligation of the anterior descending branch of the left coronary artery die or develop infarction when the implanted internal mammary artery is occluded. An internal mammary coronary artery anastomosis has been shown to be of functional value. It has relieved artificially produced coronary artery insufficiency. It is reasonable to assume on the basis of our experimental work and the pathological facts of coronary artery sclerosis that an internal mammary artery implant may be of value in the treatment of human coronary artery insufficiency.

Cases have to be carefully selected and treated by a medical-surgical team, and it is our opinion that the use of quinidine pre- and post-operatively with procaine during the operation is important in preventing ventricular fibrillation. Our first patient, we believe, develped his coronary thrombosis because of the continuous low blood pressure which was present throughout the operation. In order to bring these patients through an intra-thoracic operation without further damaging their coronary artery system,

we believe it is necessary to maintain their blood pressures above 100 mm. Hg. throughout the entire operative procedure.

The postoperative care of these cases is fundamentally a medical problem. Unlike other thoracic cases, these patients should not be moved frequently in the first few postoperative days. It would seem best to treat them postoperatively much in the same manner as a case of coronary artery thrombosis. Interpretation of postoperative electrocardiographs is difficult because of the disturbances created by the implantation of a pulsating artery into the anterior wall of the left myocardium. Clinically there has been no evidence of coronary artery thrombosis developing after operation in the two cases which have survived. It is too early to estimate results.

SUMMARY

1. The internal mammary artery can be implanted in the ventricular myocardium in man with recovery.

2. There appears to be no disturbance in cardiac function resulting from the implant procedure.

3. The internal mammary artery in man, as in the animal, was found to be completely patent 62 hours after implantation in the one fatality that occurred.

4. In spite of the burying of an open vessel in the myocardium, there was no evidence of hæmorrhage or intramural hæmatoma.

5. The last two cases appear to have been improved at the time of discharge.

We wish to express our appreciation for the counsel, criticism and continued support which have been given by Drs. Lyman Duff, Donald Webster and C. A. MacIntosh. In particular do we wish to thank Dr. G. R. Brow for his careful selection of cases for operation and for his help in their postoperative care.

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BOOKS ON ART FOR WAR-DAMAGED LIBRARIES.—In response to a Unesco appeal for art books and prints to war-damaged libraries, the San Francisco Museum of Art has sent material of this kind to Austria, Czechoslovakia, Germany, the Netherlands and Poland. Subscriptions to various American art periodicals have also been donated.—(UNESCO.)

AMNIOTIC FLUID EMBOLISM*

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SINCE the first 8 cases of amniotic fluid embolism were described by Steiner anl Lushbaugh[1] in 1941, 14 additional cases have been reported, thus justifying the authors' prophecy that this condition would be found to be an important cause of maternal mortality. They estimated its incidence to be once in every 8,000 confinements, and said it was responsible for 0.2% of all maternal obstetrical deaths. In a recent report in 1949, the same authors[13] altered their estimate of its incidence to once in every 20,000 confinements.

Because we understand so little of the mechanism of death in amniotic fluid embolism and because of the lack of prodromal symptoms, little has been accomplished in its prevention. In the present paper an additional case is reported, previous cases are reviewed, and the significance of the finding of blood incoagulability is discussed.

CASE REPORT

A 39 year old woman had had three normal, full-term deliveries in 1935, 1938 and 1946, respectively, and these infants had varied from 8 lb. 14 oz. to 7 lb. 14 oz. in weight. Three other pregnancies in 1943, in 1944 and in 1949 had terminated in abortion for unknown reasons. During hospitalization following the last abortion in 1949, she required transfusion for blood loss.

During her seventh pregnancy, the patient was carefully followed in prenatal clinic. The date of expected delivery was April 8. Physical examination revealed no abnormality. The blood Wassermann reaction was negative, and the blood group was O, Rh positive. Urinalyses revealed a faint trace of albumin on five occasions, and a faint trace of sugar on one occasion. At the last prenatal visit, 13 days before admission to hospital, the baby was active, but the fetal heart was inaudible.

The patient was admitted to hospital on April 15 (at 41 weeks) in the second stage of labour. Spontaneous rupture of the membranes had occurred half an hour previously. Labour pains came on every two to three minutes, and were of such severity that the patient had difficulty in walking from the automobile into the hospital. The cervix was well dilated, the fetal head well down in the pelvis, the presentation vertex, right occipito-posterior, and the fetal heart inaudible.

One and a half hours after admission, the patient was anæsthetized with ether and oxygen, following induction by nitrous oxide and cyclopropane. Manual rotation was performed, and a dead, 9 lb. 12½ oz. baby was delivered by low forceps, after removal of a loop of cord from around the infant's neck. The patient remained in excellent condition during this procedure.

Twenty minutes after delivery, during repair of the episiotomy, before delivery of the placenta, the patient suddenly became profoundly shocked, her blood pressure

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was unobtainable, and she was pulseless, markedly cyanosed and apnœic. There was no evidence of pulmonary œdema.

She was given coramine, methedrine, intravenous blood, plasma and normal saline, of total volume 3,150 ml. following which slight improvement occurred. A faint rapid pulse at 140/minute became palpable, but the cyanosis persisted, and the blood pressure remained unobtainable.

At this time the placenta, which had already separated, was removed manually from the vagina, and careful exploration revealed no cervical tear or uterine rupture. The blood loss during labour was not unusual, and was estimated at 500 c.c.

Despite continued treatment, her condition gradually deteriorated, and she died two and a half hours after delivery, and two hours and ten minutes after the onset of symptoms of shock. The total duration of this woman's labour was two and a half hours. Unfortunately, the character of the amniotic fluid was not noted. The clinical diagnosis made by Dr. Jocelyn Rogers was amniotic fluid embolism.

Summary of Autopsy Findings

Slight dependent pulmonary congestion; slight bilateral hydrothorax (50 c.c.); ascites (150 c.c.); retroperitoneal pelvic hæmatomata; petechiæ of peritoneum and skin of abdominal wall; small hæmorrhages into the recti abdomini, and two small fragments of retained placenta in the gravid uterus, were found at necropsy.

Gross appearance.—Both pleural cavities contained 50 c.c. of pale pink watery fluid. The lungs together weighed 450 gm. and except for slight basal congestion, were normal. The larynx, trachea, and bronchial tree were freely patent, and the pulmonary vessels contained only fluid blood.

The hæmorrhages in the pelvis were not considered excessive for the post-partum state. The cervix and cervical canal were soft and easily dilated. Except for two small, irregular fragments of retained placenta, firmly attached to the anterior wall, the uterine cavity was empty, and was lined by thick, spongy, yellow-pink tissue. Numerous, minute, superficial erosions were present in the mucosa of the cervix and cervical canal, and there was a moderate amount of sub-mucosal hæmorrhage in the cervical canal. No cervical or uterine lacerations were present. Both tubes and ovaries were normal.

Blood removed from the right ventricle during autopsy failed to clot after three days. After centrifugation, using the method described by Gross and Benz[2] no third layer separated from the blood, and no emboli were found on microscopical examination of the plasma.

Microscopic Findings

Lung.—Very many of the capillaries and arterioles were occluded by epithelial squames (Fig. 1) typical of those found in amniotic fluid, and amorphous granular debris which was found to contain abundant fat globules when frozen sections were stained with Scharlach R. Many of the small arteries were occluded by mucin. Polymorphonuclear leucocyte infiltration of these emboli had occurred in varying degree. No intra-alveolar hæmorrhage or œdema was present.

Uterus.—Similar emboli were present within the venous sinusoids of the uterus (Fig. 2).

Kidneys.—In occasional glomeruli small masses of epithelial squames obstructed the capillary loops, (Fig. 3) and the appropriate stains showed the presence also of fat and mucin.

No emboli were seen in sections of brain or heart.

Discussion

Including the present report, 23 cases of amniotic fluid embolism have now been reported: Steiner and Lushbaugh,[1] 8 cases; Lushbaugh and Steiner,[3] 2 cases; Hemmings,[4] 1 case; Gross and Benz,[2] 3 cases; Goodof,[5] 1 case; Wyatt and Goldenberg,[6] 1 case; Jennings and Stofer,[7] 1 case; Watkins,[8] 1 case; Barron, Sturley and Lindsay,[9] 2 cases; Shotton and Taylor,[10] 1 case and Steiner, Lushbaugh and Frank,[11] 1 case.

The cases up to 1948 were reviewed by Jennings and Stofer,[7] and in 1949, Shotton and Taylor[10] published a study of 15 cases. The pathogenesis of amniotic fluid embolism was effectively demonstrated by Steiner and Lushbaugh[1] in 1941, it has been well reviewed by several authors since, and at present there seems nothing further to add. The main features of all published cases, therefore, need only to be summarized briefly.

The age incidence has varied from 25 to 44 years, with an average age of 31 years. All but four patients were multiparous, and the catastrophe occurred more frequently in the second pregnancy. Labour was associated with strong, severe or tetanoid uterine contractions in over half of the cases. The duration of the mother's survival from the onset of symptoms of shock to death varied from five minutes to eight hours with an average survival time of one hour and

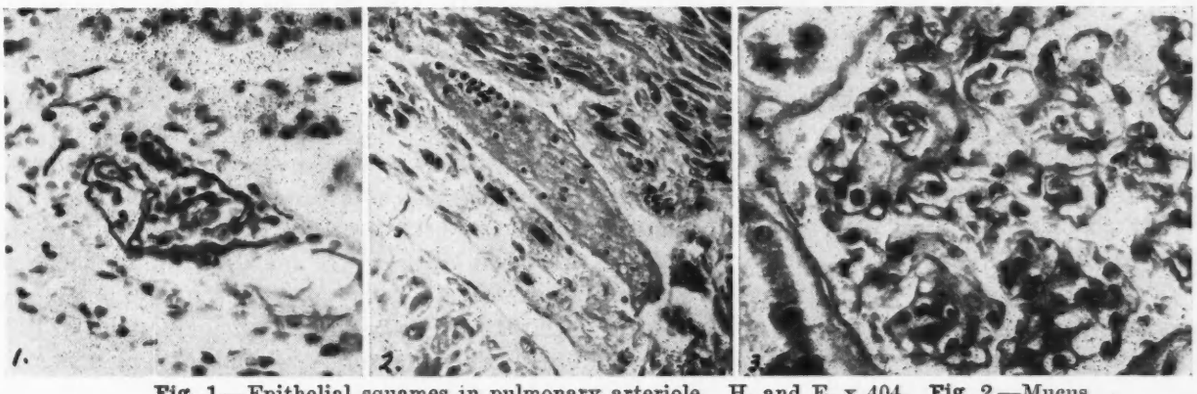

Fig. 1.—Epithelial squames in pulmonary arteriole. H. and E. x 404. **Fig. 2.**—Mucus and white cells in uterine vein. H. and E. x 96. **Fig. 3.**—Compressed squames occluding loop of renal glomerulus. H. and E. x 404.

fifty minutes. From what information there is available, 3,725 gm. was the average weight of the infant, with a variation from 3,032 to 5,568 gm. Two-thirds of the infants were dead on delivery, and of the remaining 8 infants born alive, two died of respiratory failure shortly after delivery. The duration of pregnancy, in the majority of the cases, was prolonged beyond 40 weeks. In 4 cases the amniotic fluid contained meconium, in 4 cases it contained blood, and in two cases there was hydramnios.

The gross findings at autopsy are meagre, but the histopathology is pathognomonic. Amniotic fluid emboli, which are easily seen under high magnification may be composed of one or more of four constituents. These are: desquamated epithelial cells or squames; mucin; granular eosinophilic debris, and lanugo hair. Frequently, the emboli are invaded by polymorphonuclear leucocytes to a variable extent, and these cells tend to arrange themselves in rows, presenting the so-called "combed-out" appearance. The squames, eosinophilic debris and lanugo hair, are well-known components of amniotic fluid, and arise from desquamation of the fetal skin. The mucin is a constituent of meconium, and is derived from the fetal gastro-intestinal tract. These emboli have been described occluding the small arteries, arterioles and capillaries of the lungs, the venous sinusoids of the uterus and the capillary tufts of the renal glomeruli. Previous authors have also described them in the capillaries of the heart and brain.

The pathological findings, then, are typical, but one feature to which little attention has been paid is the incoagulability of the blood. Gross and Benz[4] first drew attention to the blood incoagulability, and used it as a basis for their diagnostic test. Since then, it has been reported in only 5 cases. Because it is a finding not routinely sought at autopsy, it may be easily overlooked. To rule out the possibility that amniotic fluid itself could alter blood clotting time, we added 1 c.c. of normal amniotic fluid to 10 c.c. of whole venous blood. This mixture clotted in normal time and simultaneously with a control sample mixed with normal saline, instead of amniotic fluid.

The most likely cause of such a sudden change in the blood coagulability would seem to be an excretion of excessive heparin, and experimentally, Jacques and Waters[12] have obtained a high yield of heparin from dogs' livers in true shock, associated with sudden prolongation of clotting time. Unfortunately, blood from cases of amniotic fluid embolism has never been titrated for heparin content. Nevertheless, working on the basis that heparin might be responsible for the incoagulability of the blood, the present case was investigated further.

The mast cells of the body, situated in many regions (skin, lung, adrenals, bowel, liver), are believed to be the source of heparin, and at the present time a discharge of granules from the mast cells is considered to be evidence of a discharge of heparin into the blood and tissues. At rest, the mast cell is a large, rounded cell, the nucleus of which is concealed by a central mass of deep purple granules (Fig. 4). When mast cells become active, they throw out pseudopodia of irregular size and shape, and their granules are either discharged into the surrounding tissues, or are carried away from the nucleus, in the cytoplasm of the pseudopodia. In this active, so-called "amœboid state" the pale blue nucleus becomes visible (Fig. 5).

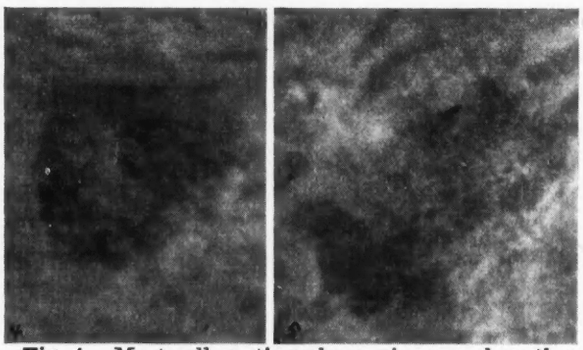

Fig. 4.—Mast cell, resting phase, pinacyanol erythrosinate. x 1,200. **Fig. 5.**—Mast cell, active amœboid phase, pinacyanol erythrosinate. x 1,200.

With the help of Dr. Sylvia Bensley, sections of skin, lungs, adrenals, bowel and liver from the present case were examined for activity of mast cells, using the pinacyanol erythosinate staining technique. There was a definite increase in the number of mast cells, particularly in the adrenals, but also in the lungs and skin. The majority of these cells were in the active, amœboid state. Thus, assuming that mast cell activity parallels heparin excretion, we have some evidence of an increase in blood heparin in the present case that may explain the blood incoagulability. We know of no evidence to suggest that heparin excretion is affected by sympathetic or parasympathetic stimulation. We are unable, therefore, to relate the phenomenon of blood incoagulability to the currently accepted theory of the mechanism of production of the clinical signs.

The cause of death in amniotic fluid embolism is a matter of great controversy. Steiner and Lushbaugh[1] produced symptoms typical of amniotic fluid embolism in rabbits and dogs by intravenous injection of the particulate matter of amniotic fluid, but were unsuccessful when they used the filtered fluid fraction. They were satisfied therefore that heterologous protein played no part in producing the sudden symptoms of asphyxia and syncope, and suggested that death might be due to nervous reflexes initiated in the lung by emboli causing cardiac depression and pulmonary vascular spasm. The lung is known to have a rich sensory nerve supply with connections to the vagal nuclei (de Takats *et al.*,[13, 14, 15] Daley *et al.*[16]). One difficulty in this interpretation is that mechanical occlusion by particulate matter other than amniotic fluid debris, and of a size sufficient to obstruct pulmonary arterioles, produces asphyxia alone (Gibbon and Churchill[17]). Syncope occurs only when the main pulmonary artery is occluded.

Most authors have therefore used the evasive term, anaphylactoid shock, in describing the symptoms of amniotic fluid embolism. This term was used by Hanzlik and Karsner[18, 19] to describe the results of injecting non-protein colloid substances intravenously into guinea pigs. This produced dyspnœa, cyanosis, trembling, apnœa, relaxation of the sphincters, spasms and death, and prolongation of blood clotting time. The clinical signs of amniotic fluid embolism in the 23 cases which have been reported are, in order of frequency, shock, cyanosis, pulmonary œdema, dyspnœa, absence of pulse, apnœa, coma, convulsions, nausea and vomiting, restlessness and chills. Hanzlik and Karsner placed most weight on the lung lesions in their guinea pigs. Thrombi and agglutinated particles were found in the small vessels. We see no reason to think that all colloid material would be arrested in the lungs and consider that there may well have been a direct action on the mid-brain nuclei. Pulmonary œdema itself, which is a common finding in amniotic fluid embolism, has recently been produced experimentally by direct stimulation of the central vagal nuclei by intracisternal injection of a fibrin-forming mixture, whole blood or particulate suspensions (Cameron and De[20]). In amniotic fluid embolism particulate protein certainly gets into the peripheral circulation, and the clue to symptoms may lie in the central nervous system.

SUMMARY

1. A case of amniotic fluid embolism is reported.

2. The number of "active" mast cells in the adrenals, lungs and skin was greater than normal, suggesting that heparin excretions may have been responsible for the blood incoagulability found at post mortem.

3. The cause of death in this condition is still obscure.

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CONSIDERATIONS IN OCCUPATIONAL AND INDUSTRIAL DERMATOSES*

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THE skin is attracting an increasing amount of attention in occupation and industry. Legislation for the protection of workers in various occupations and industries has been passed in many countries. The skin is the only organ of the body that is exposed to environmental factors, and the worker is more apt to attribute a skin disease to his work than a disease of some internal organ. As a result, more certificates of disablement have been issued for conditions of the skin than for any other compensable condition.

Guy Lane estimated that 2 to 12% of all dermatoses are of industrial origin and that the dermatoses comprise 65% of industrial disease.

* Read at the Canadian Dermatological Association meeting in Quebec City, June 9, 1950.

exclusive of accident. Schwartz found that the number is increasing and gives 70%, and he states: "in general the tendency to ascribe skin diseases to industrial environment is too great, and rigid requirements should be followed before diagnosis of the industrial dermatosis is made". Workers in dye plants are apt to attribute all cases of skin irritation to the chemicals they handle; dermatophytosis, Rhus toxicodendron poisoning, urticaria, psoriasis and even scabies have been claimed by them to be due to the chemicals which they handle.

It is the task of the dermatologist to determine whether the dermatosis has been produced by the industrial environment, or perhaps has been merely irritated by environmental factors, or has nothing to do with the patient's occupation. Methods of testing the patient's skin to materials used in industry are now available which are of great assistance in making a decision as to the rôle of occupation in production of the dermatosis. Improvement or complete relief after mere cessation of work is not necessarily a proof of industrial causation, but removal from one portion of a plant to another, if followed by relief, usually signifies elimination of irritating substances. Early recognition of occupational dermatoses at a time before a worker has become completely disabled, a timely change of occupation and prophylactic measures in the form of protective clothing and sanitary improvements, will be instrumental in combating this serious problem.

The most common industrial dermatosis is that due to external irritation belonging to the eczema-dermatitis venenata group. The eruption consists of the erythema, œdema, vesiculation, oozing and crusting syndrome, followed by scaling if the dermatosis becomes subacute. Chronicity is signalized by thickening, lichenification, fissuring and hyperpigmentation. The eruption is on the surface exposed to the irritant, usually the hands and face. Maceration by water, alkalis, and high temperature favours the production of dermatitis. The offending substance may be gaseous, liquid or solid. If the patient's skin is hypersensitive to the offending agent, the eruption is known as eczema, but if the majority of exposed persons react, it is called dermatitis venenata.

The question of sensitization is important in these eruptions. A worker may develop dermatitis on the first apparent contact with a certain substance. If the patient is hypersensitive, previous contact must be assumed although it is often difficult to determine. Usually a worker will come in contact with a substance for varying periods of time before hypersensitiveness is manifest. This incubation period varies from a few days to many years. The chief implication of the possibility of sensitization is that contact with substances with impunity for years does not mean that the individual may not become hypersensitive to them. In certain phases of industrial work, such as exposure to dust in the grinding and mixing operations in the dye industry, negroes are employed because it has been found by actual experience that they are less sensitive to skin irritation; but on the other hand the negro race, for unknown reasons, is especially subject to the development of keloids which often follow a burn, cut or other injury.

There are a number of factors which predispose to the development of eczema-dermatitis venenata; age predisposes to a certain extent, acute dermatitis is seen more often in young and new workers; chronic dermatitis is observed in older individuals. Women are more predisposed to dermatitis than men, especially during menstruation and gestation, by an increase of perspiration. The type of skin constitutes an important factor. Many factories will not employ workers with thin, white, blond or ichthyotic skin in occupations that would bring them in contact with potential irritants. Workers having thick oily skin withstand the action of fat solvents such as soap, turpentine, naphtha, benzol and carbon tetrachloride better than those with dry skin. On the other hand, in occupations where oils, greases or waxes are apt to soil the clothing, as in oil refineries, machine shops, garages, etc., we have observed that individuals with hairy arms and legs and seborrhœic skin are more likely to develop acne-like lesions and folliculitis. Another factor is perspiration. It has been observed that workers who perspire excessively are more likely to develop dermatitis from substances which are irritating only in the presence of moisture or when in solution. Excessive perspiration combined with friction tends to maceration of the skin, and this renders it less resistant to external irritants. Contact with materials which macerate the skin predispose to many eruptions, especially dermatitis. Solutions used in machining metal are soapy and macerating and in addition often contain sulfonated vegetable oils, to which some workers are hyper-

sensitive. Sweating in overheated rooms or in the summertime increases the tendency to eruptions where protective masks are in contact with the skin. Workers wear less clothing in summer which permits dust of various kinds to penetrate to the skin producing irritation. Lack of cleanliness and prolonged use of soiled clothing tends to the production of dermatoses.

An interesting observation in connection with occupational dermatitis is that in a case where the condition develops only to a mild degree soon after an individual is first exposed to a substance, the dermatitis tends to disappear after a time, even though exposure is continued. This feature is commonly known by the workers as hardening. Osborne and Jordan stated that in their opinion it is only the dermatitis produced by primary irritants, and not allergic dermatitis, which will undergo recession as a result of hardening of the skin. They explain the "hardening" process by production of hyperkeratosis which supplies additional protection. The eczematous type of eruption is distinguished by the fact that it is vesicular and diffuse. It is true that local application of substances will produce localized lesions, but even these are distinctly vesicular and diffuse throughout the affected area. When the hands and feet are involved the dorsal surface is affected first and the palmar or the plantar surface only secondarily, since the palms and soles are notoriously resistant to external irritants.

The agents that have been found to produce dermatoses in occupation will be discussed under five main headings.

1. *Mechanical agents* give rise to callosities, hygromata, tenosynovitis, cuts and abrasions, and wounds of the skin which become infected with bacteria and fungi, and can also become the site of malignant growths, of new areas of psoriasis, lichen planus, etc.

2. *Physical agents* act on the skin in various ways. High temperature causes perspiration and softening of the horny layers of the skin, and combined with friction produces intertrigo and the so-called "heat-rashes" common among workers exposed to hot areas, such as stokers and steel makers.

(*a*) Hot water softens the epithelium and renders the skin vulnerable to the action of substances such as mild alkalis that would not otherwise affect it. Dishwashing and laundry work are examples of occupations in which maceration of the skin in hot water help to cause dermatitis.

(*b*) Cold, especially when accompanied by dampness, produces pernio and frostbite, and in some hypersensitive individuals urticarial lesions have been known to develop from this cause.

(*c*) Electricity causes occasional burns from short circuits, live wires and bare wires among linesmen and others engaged in electric power production.

(*d*) Radium and roentgen-ray dermatitis, burns and cancers are occupational diseases to which radiologists and technicians are subject. The use of x-rays in the detection of flaws in metals opens up a new industrial hazard. The industrial use of radioactive substances as radium, radiothorium, mesothorium and uranium exposes workers to the action of these rays which can cause burns and cancers. They can cause dermatitis on the hands, arms, loss of nails and ulcerations leading to cancer. The principal workers exposed are x-ray machine workers, radiologists, radium and uranium miners, physicians and dentists. Ultra-violet rays are industrial hazards among electric welders where they cause eye flashes and flash burns of the skin. Prolonged exposure to solar radiation occurs among farmers, sailors and produces melanosis and some proliferative changes in the skin which, in some instances, becomes epitheliomatous in character. Blonds are more sensitive than brunettes.

3. *Chemical agents* are the predominant causes of dermatoses in the manufacturing industries. These can be divided in two groups: organic and inorganic. Each of these groups can be divided into two groups: (*a*) primary skin irritants; (*b*) sensitizers. A primary cutaneous irritant is an agent which will cause dermatitis by direct action on the normal skin at the site of contact. A cutaneous sensitizer is an agent which does not necessarily cause demonstrable cutaneous changes on first contact, but may effect such specific changes in the skin that after five to seven days followed by further contact on the same or other parts of the body dermatitis will result. The list of chemicals involved, as well as sensitizers, is very long, and may be found in the textbooks.

4. *Plants and woods.*—Many plants, especially those belonging to the Rhus family, irritate so large a percentage of people that they may be classed practically as primary irritants. The toxic properties of plants vary with the season

and the state of their growth. Many plants are photosensitizers. Woods, especially when being sandpapered and polished, have been found to be poisonous to a considerable percentage of workers.

5. *Biological agents* often cause or complicate occupational dermatoses. (*a*) Bacterial infections: folliculitis, boils, anthrax, impetigo, erysipeloid, butcher's pemphigus. (*b*) Fungi: dermatophytes, sporotrichosis, blastomycosis, monilia. (*c*) Parasites: hog itch, grain itch, ground itch and others.

Symptoms of Occupational Dermatoses

These depend in large measure on the cause and the clinical type of the lesion. In cases caused by concentrated irritants or by hypersensitivity, where the onset is sudden, the patient first notices itching of the exposed parts. This is followed by an erythema, papules, vesicles, œdema, oozing and crusting. The eruption is an acute moist eczema. The inflammation may be arrested at any of these stages. A worker may not develop symptoms for a period of time varying from a few hours to a few days after contact with the offending substance, and in certain cases of hypersensitivity, it may take years of contact to become sensitized. Some workers develop only a mild dermatitis, are able to continue working, and finally become immune. Workers who suffer a severe attack do not as often develop an immunity as those who suffer a mild attack. Immunity, if developed, generally lasts only a short time, from a week to a month, after discontinuing work. The worker who, after working without any trouble for many years, suddenly becomes sensitized to materials he formerly handled with immunity and reacts with a severe dermatitis, never becomes immune; he has lost his immunity.

In cases of exposure to mild irritants over long periods of time the early symptoms are not annoying, but consist only of a mild irritation followed by thickening of the skin and loss of elasticity. This later results in cracks and fissures, and the open sores are irritated and kept inflamed by the entrance of more of the causative agent. Ulcers result, and these heal only with difficulty while the patient is working. Such cases present the picture of a chronic fissured eczema. Dehydrating agents such as salt, sugar, and low concentrations of lime and formaldehyde may cause such lesions. Such cases are not due to allergy but to an exhaustion of the defence mechanism of the skin. Substances like mineral oil, grease, paraffin, chlorinated synthetic waxes, and coal tar pitch can not only cause dermatitis but if allowed to stay in contact with the skin for long periods will block the pores, irritate the epithelium of the skin glands and cause comedones, acnes, and cysts, which may become infected and result in folliculitis and boils.

Petroleum, unrefined paraffin, grease, tar and pitch cause epithelial proliferation which may result in keratosis, papilloma and epithelioma of the exposed parts. Tar, pitch, soot and petroleum may also cause scrotal cancers. Exposure for long periods to certain aniline derivatives such as alphanaphthylamine and benzidine may cause papillomata and carcinomata of the bladder. Arsenic is more likely to produce keratosis of the palms and soles when taken internally than by occupational exposure. Occupational mycotic infections such as ringworm of barbers and mycoses of the hands and nails of vegetable and fruit canners are not infrequent.

Diagnosis of Industrial Skin Disease

It is important to determine whether a dermatitis from which a worker is suffering is of industrial origin: (1) because it has a direct bearing on the treatment of the case and on the prevention of its recurrence; (2) because of the compensation involved; and (3) in order to determine who pays the physician's fee. The history of the dermatitis is most important. In order for the dermatitis to be considered as of possible occupational origin, it must be brought out that such a dermatitis was not present before the patient entered the occupation. It must also be shown that the dermatitis developed during the period of industrial exposure, or after a lapse of a reasonable incubation period since the cessation of exposure. This lapse should not be over a week. If the history shows that other workers similarly employed are similarly affected, then the possibility of a diagnosis of industrial dermatitis is strengthened. If the history shows that the dermatitis develops whenever the worker is at work, gets well or improves when he is away from work and again recurs when he returns to work, then the history itself establishes a definite cause and the relation between occupation and the dermatitis.

The site of the eruption is also important.

In examining patients, they should be completely divested of clothing. Occupational dermatitis usually begins on the exposed parts, the hands, the fingers and the forearms if the offending material is a solid or a liquid; and on the face and neck if it is a vapour. The covered parts of the body may also be affected if fumes or vapours penetrate the clothing, or if the clothing is not frequently washed and becomes saturated with irritant chemicals. The dermatitis is also often found at points of friction on the body. Sometimes a dermatitis of undoubted occupational origin may become generalized. This occurs when the irritant is one to which the worker has developed a high degree of sensitivity.

An industrial dermatitis of the acute eczematoid type begins as an erythema, followed by papules and vesicles and, when the vesicles break, by an oozing and crusting no matter what irritant is the cause. Occupational mycotic infections, such as ringworm among bath attendants, barbers, beauty parlor operators, and yeast infections among cannery workers usually occur on the hands, but the appearance is not different from that of ringworm or yeast infections of non-industrial origin. Paronychia and onycholysis are common lesions among fruit and vegetable canners. Certain tar compounds also cause acne-like lesions on the exposed parts. Oils cause folliculitis and boils, especially on the hairy portions of the body. Paraffin, grease and tar cause keratoses to develop on the hands and forearms, and these keratoses occasionally become malignant. Keratotic lesions and excessive pigmentation around the face and the neck may be occupational among workers exposed to the sun such as farmers and sailors. Occupational dermatitis must be differentiated from such diseases as seborrhœic dermatitis, fungus infections, lichen planus, impetigo contagiosa, pityriasis rosea, erythema multiforme, drug eruptions, neuro-dermatitis and dermatitis due to contact with irritants met with outside of the place of occupation.

The greatest difficulty in the differential diagnosis is presented by dermatitis due to contact with substances met with outside of the place of occupation. In these cases the lesions are similar in appearance and site, and only the most careful consideration of all the facts can lead to a correct etiology. It is here that the patch test is of greatest value. It is now universally accepted that the patch test, if properly performed and interpreted, is a valuable diagnostic procedure. Its value in preventing possible outbreaks of dermatitis from the use of materials containing new chemicals before they are put into general use is just becoming recognized.

TREATMENT

The therapy of industrial dermatoses has an advantage over that of general dermatology due to the fact that in most instances the offending agent is known, and by its elimination together with the use of soothing applications an immediate cessation of symptoms can be brought about. The common way in which the skin reacts to various irritants gives the industrial dermatologist a useful clue to the causative factors. The medical therapy of skin disease must be undertaken with caution, as in many cases treatments only make the dermatitis worse. In applying medication to the lesions only the mildest ointments and lotions should be used such as boric acid or calamine. Strong ointments are apt to irritate the skin and cause more dermatitis. Sometimes eruptions treated with mercury, sulphur, and many proprietary ointments or sulfonamides are quickly changed in to severe dermatitis. Diagnosis and treatment by doctors unfamiliar with skin disease may also be disastrous, as many ordinary affections are mistaken for industrial dermatitis.

We believe that most occupational dermatoses are exacerbated either by irritating applications, scratching, frictions from clothing, secondary infections, and occasionally by malingering, before the patient comes for medical treatment.

In acute dermatitis, presenting a vesicular eruption on an erythematous base, with œdema, etc., the continuous application of cold wet dressings is advised with the following solutions: Boric acid solution, Burrow's solution, 10%; potassium permanganate 1 to 5,000 solution; calamine lotion and zinc, etc. When the skin becomes dry and feels tight boric acid ointment, zinc oxide salve, petrolatum, olive oil, albolene, etc., should be applied. When the dermatitis is extensive, relief from pruritus may be secured by warm baths containing corn starch, bran, oatmeal, salt, etc.

Complications such as infections, erosions and ulcerations must be treated with antiseptics. Infected follicles and furuncles should be opened daily, crusts removed, and the lesions washed

with any mild antiseptic solution followed by painting with an aqueous solution of brilliant green, 0.5%, or gentian violet, 5%, in alcoholic solution. If the infection is impetiginous, ointments with ammoniated mercury or streptomycin; these ointments should be applied twice daily after washing.

When the skin has become dry, scaly, fissured and crusted oily substances are indicated. Gauze soaked in olive oil, mineral oil, castor oil, solid albolene, unscented cold cream, calamine liniment, soft paste and watery paste should be used. It is best to bandage the parts well with rubber tissue, cellophane, or oiled silk and leave on for a few days at a time. Where eczematization with lichenification occurs, counter-irritants and reducing agents such as crude coal tar ointments, salycylic acid 10% in oil, chrysarobin 5% in ointment have been proved useful. Roentgen ray therapy, however, has shown good results in this type of eruption. Alpine and infra-red rays seem to have a very limited use.

In some factories where there is a hazard from alkali burns as in the manufacture of viscose, a weak solution of acetic acid is kept at hand so that it can be applied immediately to the parts which have come in contact with the alkali. Ulcers resulting from such corrossives as chromic acid and chromates, zinc chloride and fluoride are best treated by thorough curetting of the base, followed by aseptic dressings. It has been found that most skins are sensitive to analgesic agents and some to menthol or phenol, so they can be omitted. The best treatment for occupational or industrial eruptions is prophylaxis. Schwartz repeatedly emphasized the value of bodily cleanliness, and the wearing of clean work clothes daily. It is suggested that two locker rooms be available, the first for street clothes and the second for work clothes. After removing the work clothes, he takes a shower bath before dressing to go home. Some industrial organizations do patch tests on prospective employees and eliminate those who show positive reactions. The hands and face can be washed or rinsed frequently with adequate cleansing agents. Special protective coverings are useful such as cotton gloves, boots and masks. Lubrication of the skin is of great assistance; lanoline, eucerin and aquaphore are very good.

These days, with the importance and the expense of industrialization, it is important that the dermatologists should be well informed.

BARRIERS TO SURGERY IN MITRAL STENOSIS*

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MITRAL stenosis is a common condition. Since cardiac surgery has advanced so rapidly in recent years it is only natural that it should continue to challenge the courageous surgeon. This in spite of the fact that it involves working with diseased tissue on the left side of the heart, in contrast to the handling of normal tissue on the right half of the circulation in cases with congenital heart disease where results have been so good.

In 1941 we wondered if surgical advantage could be taken of the normal movement of the mitral valve. We thought that a sheathed Gigli saw inserted through the left auricular appendage and brought out through the apex of the left ventricle might provide a taut blade against which the valve would "cut itself" from its orifice to its base without loss of valve substance.

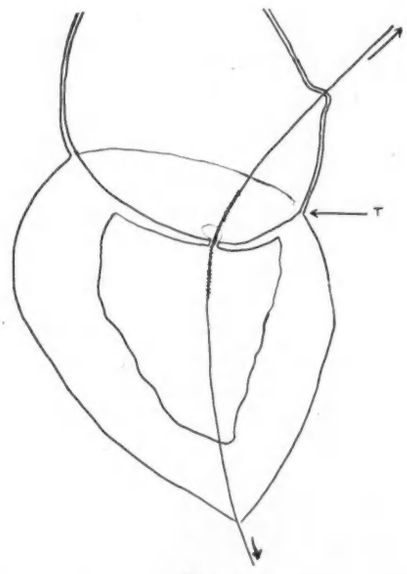

Fig. 1

We found that the dog's heart was not disturbed by this procedure and in 1948 we chose a human case with mitral disease for valve section. However the difficulties began to multiply once we embarked on the human

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experiment and our views began to change after eight years of surgical experimentation coupled with clinical and pathological studies.

CLINICAL WARNINGS

In spite of our knowledge of this condition the exact diagnosis of so-called "tight" mitral stenosis—which alone is surgical—is not always easy.

The presence of significant mitral insufficiency rules against surgery. We presume that it exists when there is more than a grade two systolic murmur at the apex. It should be suspected if the pulmonic second sound is not increased. Mitral regurgitation gives only a systolic increase of pressure in the pulmonary veins. With mitral obstruction there is systolic and diastolic overloading of the pulmonary circuit and therefore the second pulmonic sound should always be increased with stenosis.

If the auricle on fluoroscopy or electrokymography shows a true systolic pulsation we can accept this as proof of a significant amount of mitral regurgitation.

In theory, the camera-shutter type of action in a perfectly sectioned pliable mitral valve should tend to decrease the element of mitral insufficiency postoperatively. However, in actual practice it is unlikely that this ideal effect has been achieved.

The left auricle is grossly dilated in some patients with mitral disease. This dilatation is due to atrophy and fibrosis of the auricular muscle, which becomes over-distended by the inflow of blood from the pulmonary veins plus that which regurgitates from an active left ventricle. A review of the literature shows that about a half of all the examples of marked dilatation of the left auricle have been associated with organic mitral insufficiency rather than mitral stenosis. Perhaps in the cases where mitral stenosis was observed at post-mortem examination there may have been a functional mitral regurgitation through the rigid orifice. It is rather hard to understand why every case with mitral stenosis would not have some associated regurgitation and since this regurgitation transmits the high pressure of the left ventricle to the passive left auricle it may be reasonable to explain gross enlargement of the auricle on this basis.

If we therefore accept a large auricle as evidence of obvious or functional mitral insufficiency we shall not subject such patients to mitral valvulotomy since even in the best hands this operation is likely to be associated with some additional regurgitation. It is an arbitrary stand that must be taken here but we suggest that in the present state of our knowledge a left auricle which is enlarged enough to form a part of the right heart border in the antero-posterior view should be accepted as evidence ruling against surgery.

Carrying this point further we would say that no grossly enlarged heart in mitral stenosis should be operated on since such cases (in the absence of associated valvular disease) are always explained by a large left auricle with or without associated right heart dilatation.

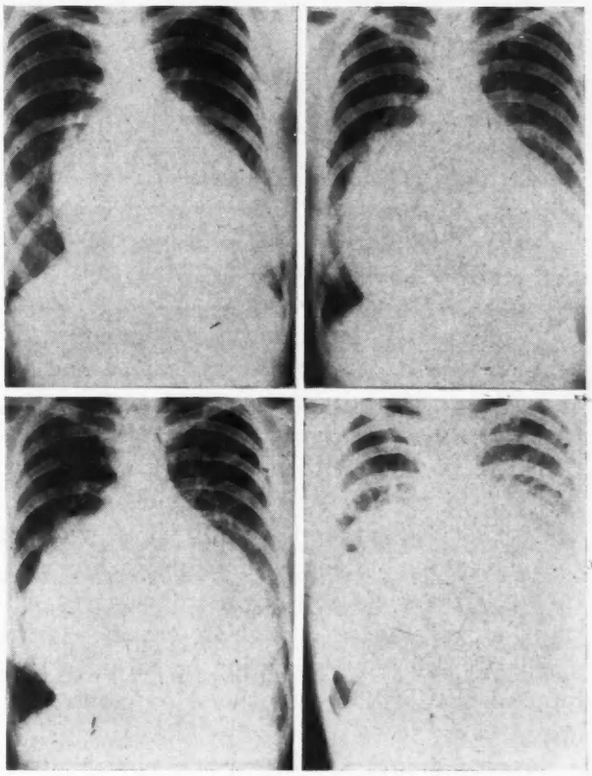

Fig. 2

A 24 year old woman was followed from 1944 to 1948 during which time the left auricle increased as shown until it touched the right side of the thorax. In addition to a marked mid-diastolic thrill a definite systolic thrill was noted. Every cardiologist after thorough examination diagnosed mitral stenosis. Her condition deteriorated. Surgery was agreed to. At operation the pressure in the left auricle was 480 mm. of water. Death occurred during instrumentation of the mitral area from ventricular fibrillation before the valve was cut. At autopsy there was no stenosis and the mitral orifice admitted three fingers easily. We were all mistaken in this case. We should have diagnosed mitral disease with insufficiency predominating.

About 10% of stenotic valves contain so much calcium that they may be visualized by fluoro-

scopy. A valve so calcified, even if it could be sectioned or fractured, would not likely open adequately in diastole before the auricular pressure. And it seems to us that the postoperative reaction in such a valve would be great enough to invite thrombosis.

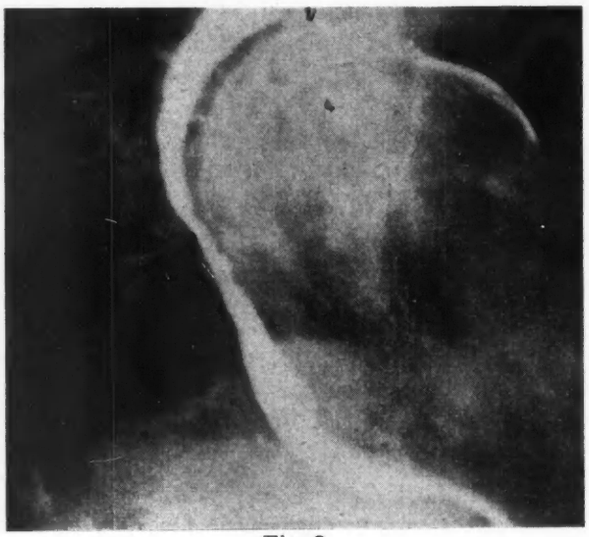

Fig. 3

X-ray films of the left auricle may show calcium in its wall. Such calcium means gross auricular disease with co-existing thrombus and it therefore excludes surgery. At operation the surgeon should palpate the appendage and the auricle very carefully for evidence of clot. This finding rules out surgery. While mitral operations can be done without too high a mortality it seems to us that such surgery should carry with it at least the possibility of a five-year survival. Any patient with palpable thrombus in the left auricle at the time of surgery will stand a real chance of embolic trouble during or shortly following operation or within a time interval that will be too short to justify such heavy work.

Surgery should not be considered if there is evidence of active rheumatic or bacterial endocarditis or significant co-existing valvular disease.

THE IDEAL VALVE FOR SURGERY

After a review of our own fresh post-mortem cases, a study of the human mitral stenotic heart perfused as a Gad physiological preparation before and after valve section, and a study of many of the larger museum collections, we have formed the opinion that the ideal valve for surgery is one that is transparent, and pliable when it is cut or torn. This pliability is very necessary if a tent-flap type of action is to occur during diastole. After viewing and palpating all the available specimens with a real surgical enthusiasm we have come to admit that not more than one case in four or five of button-hole or "tight" mitral stenosis presents this pliability.

The character of the valve cannot be predetermined by assessment of factors such as age, sex, heart sounds, size of the left auricle, or rhythm. Therefore we doubt if surgical attack is likely to accomplish anything worthwhile in perhaps 75% of the cases selected for operation because of unsuitability of the valve.

This suggests the need for combined studies at the autopsy table by cardiologists, surgeons, and pathologists who will ask themselves as they view each specimen whether anything worthwhile could have been achieved by an adequate section. Perhaps if we all do this it may be possible to decide how reasonable the surgical undertaking is from a survey of a large volume of material. At the present time there is no such collection of information that we know about.

VENTRICULAR FIBRILLATION

Whenever instrumentation of the left auricle or ventricle is carried out, the risk of ventricular fibrillation wil be encountered. Even with quinidine preoperatively, digitalis, procaine, adrenalin, and electro-shock used with the idea of defibrillating the ventricles, it is probable that this danger will exist in from 5 to 10% of mitral cases operated on. Its onset should warn the surgeon to stop at once, since an occasional case has reverted spontaneously.

HEALING OF THE SECTIONED VALVE

After section of the anterior leaf of the mitral valve in the dog the cut edges retract from each other and there is no contact between the margins of the incision with ventricular systole. Even here our animals developed thrombus along the edges of the incision in the valve.

In human mitral stenosis the cut edges (usually calcium containing) will continue to traumatize each other following surgery whether this involves the posterior cusp or the commissures. Such trauma may encourage thrombus formation. Dr. Bailey who has mentioned the point of healing particularly, reported no evidence of healing two months after an adequate valve section.

We had hoped that endothelium would grow over the cut surfaces of the sectioned valve as it does after an arterial anastomosis. Our pathologist Dr. John Fisher has studied the mitral valves from three of our animals following section with great care after survival times of six months, twelve months, and eighteen months. He found that the endothelium had not advanced over the lines of section or even over the cut ends of chordæ tendinæ in any of the three valves.

Certainly the process of healing after section in the human valve and in the experimental animal must be studied more thoroughly since a bare area in the left heart will always be a serious threat to the patient's future.

THROMBO-EMBOLIC DANGERS

These are very real and all too frequent. Dr. Levine found that 49% of fatal mitral cases with a fibrillation history showed intra-cardiac thrombi at post-mortem.

Thrombi may occur in the appendage, in the auricle, or on an ulcer that is often found on the anterior cusp. Because surgical approaches usually cross these areas it would be impossible to avoid loosening a thrombus en route. We try to prevent embolism in the operating room by asking the anæsthetist to compress both carotid arteries whenever the left auricle is being handled and when instrumentation of the heart is being carried out. Particles of clot or valve are in this way shunted away from the brain.

Postoperative thrombus may form along the line of section in the valve or over the suture line at the base of the appendage, and from these sites emboli may arise with unhappy results.

Since thrombosis after operation is so likely it does seem reasonable to try to prevent it in this period with anti-coagulants. However, their use in the most experienced hands has invited bleeding and most surgeons have abandoned them.

PROGNOSIS IN MITRAL STENOSIS

The greatest barrier is the choice of the time for operation in any given case. Between 1940 and 1944 we selected 13 cases which we listed for surgery as soon as we felt it could be done with reasonable safety. Eight of these 13 patients are still living and active without surgery. Two had evidence of marked pulmonary hypertension as shown by dilated pulmonary arteries in the x-rays and by Graham-Steell murmurs. One of these two is still living and the other one died this year with massive thrombosis of the left auricle. Only one of the five fatal cases had a valve which might have been sectioned profitably and that patient died with a brain embolism.

It is certainly a fact that remissions and exacerbations do occur in the course of mitral disease, for reasons that are not easy to explain. We should therefore move slowly toward surgery unless the whole clinical picture is becoming worse. We do not subscribe to the belief that there is such a thing as prophylactic mitral valve surgery done before the valve has reached its terminal phase of scarring.

THE SURGICAL OBJECTIVE

We should remember that given a suitable valve to section there will always be some uncertainty about the placement of the incision or tear in the valve at operation. The measure of surgical success is being estimated by postoperative catheter studies. The blood pressure in the pulmonary artery before and after valve surgery as determined by the catheter method is influenced by at least five factors. This procedure is therefore perhaps not sufficiently accurate for use in estimating improvement following operation.

The real surgical objective as we see it is a reduction of the blood pressure in the left auricle as measured in the operating room with a saline manometer. It seems fair to draw attention to the fact that this objective has not been attained in more than a very few of the reported cases. Certainly the pressure has never been reduced to a normal, which is about 60 mm. of water. Because of this fact such surgery may be palliative but it cannot be called curative in any sense of the term. Unless the surgeon can demonstrate a substantial reduction in the pressure in the left auricle at the end of his work, it is unlikely that the operation will account for any lasting gain to the patient.

SUMMARY

Even with these discouraging features there are undoubtedly some cases with mitral stenosis without marked enlargement and with evidence of severe pulmonary hypertension which can be helped by an adequate section done at a certain time.

However, when we consider the natural course of this disease, the unsuitability of most mitral valves for surgery, the uncertainty of healing, the thrombo-embolic dangers, and the hazards to be faced at operation, the number in whom prolonged improvement will follow surgery must of necessity be very small.

We think that by considering some of the points mentioned, surgery will not be attempted so frequently in patients where it cannot accomplish anything worthwhile.

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CLINICAL RESULTS OF PREFRONTAL LOBOTOMY (Leucotomy)*

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THIS is a report on the results of leucotomy in a series of 38 psychotic patients of the Ontario Hospital at London, who were operated on between May, 1943, and the end of 1949.

No attempt is made here to describe the operative procedure devised by Egas Moniz,[1] and brought to this continent by Freeman and Watts,[2] other than to state that all but one of the operations were performed by Dr. K. G. McKenzie[3] or Dr. E. H. Botterell in the neurological service of the Toronto General Hospital, using their own special technique. Patients were selected by the staff of the Ontario Hospital at London, and were transferred to the Toronto Psychiatric Hospital for immediate preoperative and postoperative care. Their suitability for the operation was concurred in by the neurosurgeon and the staff of the Toronto Psychiatric Hospital.

Table I shows the number of patients operated on in each of these seven years.

TABLE I.

PREFRONTAL LOBOTOMIES

1943	4
1944	2
1945	1
1946	1
1947	0
1948	21
1949	9
Total	38

Patients were selected from the following groups: (*a*) manic-depressives (including involutional melancholias) who had failed to recover on the usual therapies, including electro-convulsive therapy (E.C.T.); (*b*) manic-depressives who were unable to hold their recovery, as evidenced by repeated relapses; (*c*) schizophrenics, who had failed to respond to the usual therapies, including E.C.T. and, in some cases, I.C.T. (insulin coma therapy); (*d*) a small number of patients who did not fall into the foregoing categories, but who had not responded to other therapies and were either very difficult nursing problems or had some hope of improvement by leucotomy.

TABLE II.

CLINICAL RESULTS FROM LOBOTOMY
1943 — 1949

		A	*B*	*C*	*D*	*E*
Manic-depressive (including involution melancholia)	23	13	7	1	2	0
Schizophrenia	12	1	5	0	1	5
Others	3	0	0	0	0	3
Total	38	14	12	1	3	8

Table II summarizes the numbers of patients in the three diagnostic groupings and the clinical results, using a classification devised on the lobotomy service of the Westminster Hospital (D.V.A.). In this classification there are five grades of response: A—complete clinical recovery and at home. B—good clinical and social improvement and at home. C—modest improvement but able to be cared for at home or in an approved boarding home under super-

* Read at a meeting of the London Academy of Medicine, September 28, 1950.

No.	*Age on admission*	*Date of admission*	*Diagnosis*	*Date of lobotomy*	*Length hospital before operation*	*Date of leaving hospital*	*Length hospital after operation*	*Present location*	*Grade of recovery*
1	48	18/10/40	Schiz.	27/5/43	31 months			Hospital	E
2	50	3/2/39	Man. Dep. agitated	23/6/43	52 months	9/8/43	2 months	Died 1950 cancer of stomach	A
3	52	15/9/38	Man. Dep. agitated	4/10/43	61 months	8/11/43	1 month	Died 1949 cancer of breast	B
4	52	25/9/41	Man. Dep. agitated	18/11/43	26 months	18/12/43	1 month	Home	A
5	50	7/7/39	Man. Dep. depressed	3/5/44	58 months	20/1/45	8 months	Home	A
6	50	6/3/43	Man. Dep. agitated and fearful	2/11/44	20 months	20/12/44	1 month	Home	A
7	31	5/1/40	Schiz. (male)	11/4/45 repeated 7/10/48	63 months			Hospital	E
8	34	19/11/42	Schiz.	22/10/46	47 months	15/11/47	11 months	Home	B
9	45	27/8/46	Man. Dep. both Excit. and Dep.	12/4/48	19 months	19/7/48	3 months	Boarding home	B
10	31	16/12/42	Man. Dep. Dep.	29/4/48	64 months	23/6/48	2 months	Home	A
11	41	26/5/45	Man. Dep. schizoid features	6/5/48	36 months	18/7/48	2 months	Home	B
12	64	18/12/46	Man. Dep. agitated	13/5/48	17 months	23/6/48	1 month	Home	B
13	46	21/3/46	Man. Dep. Dep.	20/5/48	26 months	8/7/48	2 months	Home	A
14	53	18/7/42	Man. Dep. Dep.	3/6/48	71 months	17/2/49	8 months	Boarding home	C
15	58	11/1/43	Man. Dep. agitated	22/7/48 unilateral	66 months	3/11/48	4 months	Home	A
16	48	16/10/46	Man. Dep. agitated	17/6/48	20 months	29/8/48	2 months	Home	B
17	31	15/3/46	Man. Dep. (both)	24/6/48	27 months	11/9/48	3 months	Home	A
18	28	13/4/38	Schiz.	8/7/48	123 months	21/8/48	1 month	Home	B
19	54	15/5/46	Man. Dep. manic	5/8/48	27 months	29/9/48	1 month	Home	A
20	24	2/6/37	Schiz.	15/7/48	133 months			Hospital	E
21	23	8/2/46	Schiz.	29/7/48	29 months	6/3/49	7 months	Home	A
22	49	6/3/47	Man. Dep. manic	26/8/48	18 months	24/10/48	2 months	Home	B
23	19	16/12/44	Schiz.	9/9/48	45 months	22/12/48	3 months	Home	B
24	56	23/6/39	Man. Dep. Dep.	23/9/48	111 months			Hospital	D
25	55	30/10/45	Hypochondriasis	20/9/48	36 months			Hospital	E
26	23	31/8/47	Schiz.	25/10/48	14 months	18/6/49	8 months	Home	B
27	40	28/10/46	Man. Dep. manic	18/11/48	25 months	13/3/49	4 months	Home	A
28	64	25/11/47	Undiag. (Schiz.?)	9/12/48	12 months			Hospital	E
29	40	10/6/38	Schiz.	25/11/48	125 months			Hospital	E
30	15	25/2/46	Man. Dep. manic	13/1/49	36 months	5/6/49	5 months	Home	A
31	50	30/6/45	Man. Dep. agitated	27/1/49	43 months	30/5/49	4 months	Home	B
32	30	8/11/46	Schiz.	3/2/49	27 months			Hospital	E
33	50	3/9/46	Man. Dep. manic	15/2/49	29 months	17/6/49	4 months	Home	A
34	27	17/11/45	Defective	17/3/49	40 months			Hospital	E
35	23	19/7/44	Schiz.	24/3/49	56 months	24/12/49	9 months	Home	B
36	29	2/8/37	Schiz.	30/5/49	142 months			Hospital	D
37	38	5/1/39	Invol. Mel.	22/9/49	130 months			Hospital	D
38	54	13/5/36	Man. Dep. manic	10/11/49	42 months	4/5/50	6 months	Home	A

vision. D—symptomatic improvement but still requiring hospital care. E—unimproved and still requiring hospital care.

It will be noted that of the 38 patients who were leucotomized, 27 were able to leave the hospital. The results were spectacularly good in the manic-depressive group, 21 out of 23 being able to leave the hospital, 13 of them as grade A recoveries.

In the schizophrenic group, 6 of the 12 operated on were able to leave hospital, only 1 as grade A, 5 as grade B. Considering the relative malignancy of schizophrenia and that these patients had failed to respond to all other treatments, we consider this a very gratifying result. It should be stated, too, that the average period of treatment in the Ontario Hospital, before operation, for the 27 patients who were able to leave, was 40 months. By contrast, it is equally noteworthy that the same 27 patients required an average of only 4 months postoperative convalescence in hospital.

There were no deaths resulting from the operation. One patient has had as a sequel to the operation periodic epileptiform convulsions which are controlled by appropriate medication.

All patients with one exception had the bilateral operation. The one patient who had a unilateral operation was in the manic-depressive group and made a grade A recovery. One of the schizophrenic patients who did not recover following his leucotomy was operated on again but without the slightest improvement, remaining in group E.

The one patient who has had to return to hospital attained only a C grade after her leucotomy in 1943 and did not have the advantage of good home supervision. She subsequently died of cancer of the breast. One other patient who had her operation in 1943 has recently died of cancer of the stomach. All other patients in this series are living.

No patient has been made mentally worse by the operation, and the intellectual level of the recovered group (grade A) is considered by relatives to be just as high as prior to the onset of the illness. In not a few of this group the general personality has apparently been improved, with less tension and anxiety, freedom from psychic pain and resulting increase in happiness. Most of the recovered and greatly improved groups (A and B) have gained 30 to 50 pounds in weight. A few patients (in the B group) have shown a diminution of initiative.

Table III (on previous page) shows certain data about each patient in the complete series. All these patients were females except No. 7.

CONCLUSIONS

Leucotomy has permitted 27 of 38 mentally ill patients to leave hospital. This group had averaged 40 months in hospital before operation and had failed to respond adequately to all other treatments. The value of leucotomy in carefully selected patients has been well demonstrated.

The authors desire to express their appreciation to Dr. K. G. McKenzie and Dr. E. H. Botterell for the actual performance of the operations, and to Dr. A. B. Stokes and the staff of the Toronto Psychiatric Hospital for their excellent co-operation and assistance.

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HYPERTENSION IN TWO CASES OF RENAL ARTERY OCCLUSION*

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PERMANENT hypertension has been produced in animals by Goldblatt[1] through the means of clamping the renal arteries, thereby reducing the blood flow through the kidneys. Because the two following cases of occlusion of the orifices of the renal arteries simulate so closely the experimental clamp, it was thought to be of interest to report them in detail.

CASE 1

Miss C.C., a 27-year old factory girl was born in Montreal and lived there all her life. She could not recall any childhood illnesses, and denied measles, mumps, diphtheria, scarlet fever, tonsillitis or sore throats. In April, 1935, she underwent an appendectomy at which time the blood pressure was reported as 160/110. The urine at that time showed a trace of albumin. She made a satisfactory recovery, but shortly after operation she noticed swelling of her feet after the day's work and puffy eyes in the morning. Three years later—in June 1938—during a routine medical examination, the blood pressure was found to be extremely high and she was admitted to The Montreal General Hospital.

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On examination she was fairly well developed, but with moderate pallor. The resting pulse was 100 and regular. The radial artery wall was palpable. The blood pressure was 210/130. The heart was not enlarged clinically and the fundi were reported as normal. There was slight œdema of the legs.

Laboratory examination.—Nine urinalyses repeatedly showed albumin 2 plus, and on one occasion a few hyaline casts were seen. Anæmia was present, the red blood count being 2,790,000 and Hgb. 55% (7.5 gm.). The blood Wassermann reaction was negative. The electrocardiograph was normal. The kidney function tests revealed the presence of nitrogen retention. The blood urea nitrogen was 50 mgm. %, and the creatinine 2.84 mgm. %. The urea concentration factor was 7 (normal 50).

On bed rest and low protein diet there was little change in her condition. She was discharged home in October, 1938, but she was never able to resume her work and steadily lost weight and strength. She was troubled with intermittent headaches, vomiting, diarrhœa and drowsiness. Finally, in September, 1939, after a severe nose bleed, she was readmitted. This time uræmia was manifest, with the blood urea nitrogen 198 mgm. % and the creatinine 11.11 mgm. %. She was semi-comatose and exhibited Kussmaul breathing. The heart was clinically enlarged. The blood pressure was 175/100. The red blood cells were 2,400,000 and Hgb. 52%. She died 24 hours after admission.

Autopsy.—Apart from the major lesions described below the pertinent findings in this well developed, but thin body, 58 inches long, was moderate hypertrophy of the heart, which weighed 340 gm., and its left ventricular wall was 2 cm. thick. There was bilateral terminal broncho-pneumonia, intestinal congestion, 60 c.c. of ascitic fluid and chronic cystitis.

After evisceration *en masse* the aorta was opened posteriorly. It was found to be quite normal except at the orifices of the renal arteries (Fig. 1). Here, fleshy, lip-like structures entirely filled the entrance to the renal arteries. They were attached to half the circumference of the orifices. About the free borders of the plugs there were crescentic slits which led into the renal arteries. The fleshy lips could be raised, exposing the arterial lumina. The renal arteries beyond were quite normal.

The kidneys were contracted (Fig. 2), each weighing 50 gm. The capsules stripped freely exposing a red granular surface. The pelves and ureters were normal. On section there was obvious marked reduction of the cortices with loss of normal vascular markings.

Microscopic examination of the renal artery orifices (Fig. 3) shows that the fleshy lip is composed of loose textured intimal connective tissue thickening with a smooth, intact surface. Deep in its substance there is a small, oval shaped area of necrosis containing many rounded clear spaces suggestive of the presence of fat. In the underlying media there is a proliferation of vasa vasorum which extends into a small area of granulation tissue at the base of the necrotic zone. The appearance of these lesions is not that of atherosclerosis but rather a localized hyperplasia of the subendothelial connective tissue producing the soft fleshy nodules.

The kidneys show an extensive patchy atrophy and collapse of tubules with condensation of the supporting stroma alternating with areas of markedly dilated tubules. Many glomeruli contain more or less blood in the capillaries and in some it is conglutinated. Others show all stages of fatty degeneration and hyalinization to complete sclerosis. The tubules in the contracted zones are collapsed and atrophied. In the dilated tubules the epithelium shows various degrees of atrophy, and albuminous and granular casts are seen in the lumina. A few tubules contain red blood cells and leucocytes. The arteries are widely patent and show only a minimal degree of intimal thickening. The arterioles are patent. Their walls are slightly thickened and hyalin-

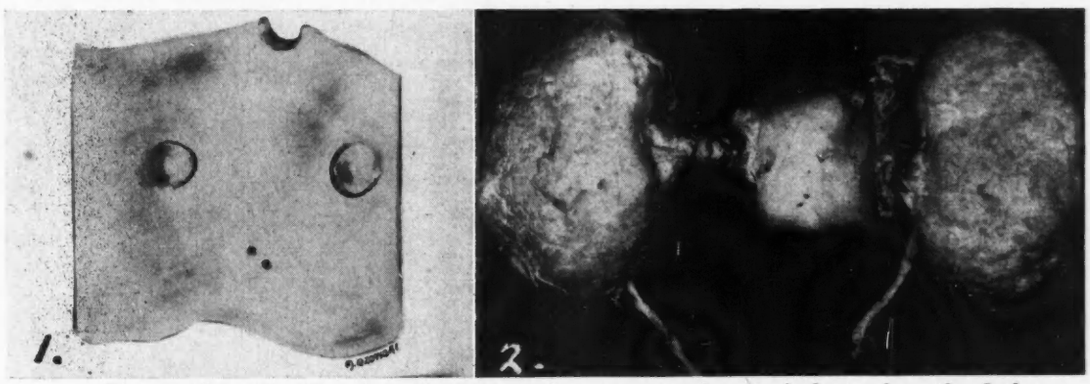
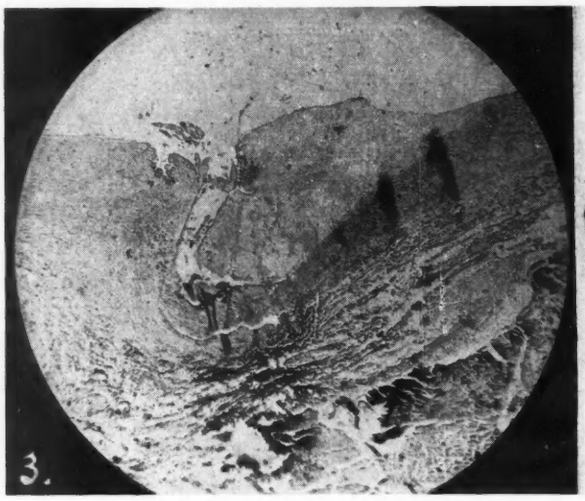


Fig. 1. (Case 1).—Drawing of segment of aorta, opened posteriorly to show the fleshy bodies obstructing orifices of renal arteries. Part of the orifice of the superior mesenteric artery is seen at upper margin and the two spermatic artery orifices are below the renal artery level. **Fig. 2.** (Case 1).—Showing the granular contracted kidneys with segment of aorta.



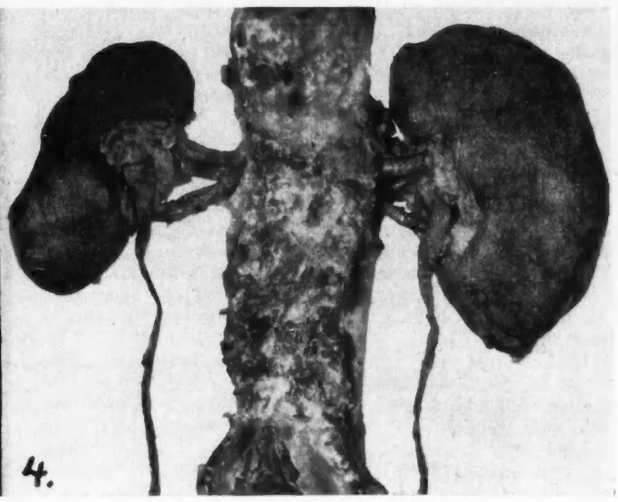


Fig. 3. (Case 1).—Photomicrograph of section through the orifice of renal artery. Note the fleshy lip almost filling the orifice. The space between the free margin of the lip and the artery wall is the result of shrinkage from fixation. **Fig. 4.** (Case 2).—Abdominal aorta, opened posteriorly, and kidneys. Note the double renal arteries on both sides, the marked atherosclerosis of the aorta obscuring the four renal artery orifices, the enlarged right kidney and the small left kidney markedly shrunken in the upper half.

ized and by scarlet red stains are shown to be intensely infiltrated with fat. The intertubular vessels are engorged with blood. Throughout the collapsed areas of the cortex and particularly marked in the boundary zone, there is a disseminated lymphocytic infiltration.

Examination of sections of other organs and muscles reveal no arteriolar lesions other than a moderate degree of hyaline swelling in those of the spleen.

CASE 2

Mr. G.A., an insurance broker, aged 50 years, was admitted to the Montreal General Hospital by ambulance after four convulsions during the previous twenty-four hours. The personal history was quite negative and two years previously his blood pressure and heart were reported as normal. The family history revealed that his mother had died of heart trouble and diabetes, while one brother died of a cerebral hæmorrhage. The history of his present illness dated back only three months, when hypertension was first discovered during the course of an insurance examination. For two months prior to admission there was a pounding headache, insomnia, moderate loss of weight and anorexia. There were neither visual disturbances nor syncope. He had been placed on rest, low salt diet, and sedation without any improvement. At 3.30 a.m., on the day of admission, he suffered from a generalized convulsion, became incontinent of urine and fell out of bed. He was given phenobarbital gr. 2, but had three more convulsions of a similar nature. After the last one he went into a state of semi-coma and was in this condition on admission.

The blood pressure on admission was 210/130. The reflexes were normal. Two days later a venesection of 250 c.c. was done with little effect on the blood pressure. The sensorium cleared but he was still disorientated. Three days later a second phlebotomy was carried out with no fall in blood pressure, and he became mentally dull, drowsy and developed polydipsia and incontinence of urine. A lumbar puncture at this time revealed a clear, colourless spinal fluid at a pressure of 180 mm. of water, with only 1 polymorphonuclear cell per c.mm. and containing 150 mgm. % of glucose and 580 gm. % of chloride. The coma gradually deepened. The blood pressure remained high, varying from 225 to 250 systolic and 120 to 140 diastolic.

Sixteen days after admission, the respiration suddenly became laboured, the pulse and blood pressure unobtainable, and he died within a few minutes.

Laboratory findings.—Repeated urinalyses revealed a variation of specific gravity from 1.015 to 1.023 and 100 to 200 mgm. of albumin. There was no glycosuria. The sediments presented occasional pus cells, rare granular casts but no red blood cells. The blood and spinal fluid Wassermann reactions were negative. There was no anæmia. The initial blood urea nitrogen was 38 mgm. % and a non-fasting blood sugar of 270 mgm. %. Four days later the examinations repeated showed a normal blood urea nitrogen of 17 mgm. % and a fasting blood sugar of 130 mgm. %. The renal test meal indicated a maximum variation of specific gravity of 6 points. A radiograph of the skull was non-contributory.

Autopsy.—Permission to open the head was not obtained. The heart weighed 370 gm. and showed slight calcification at the base of the aortic valve and a spotty atherosclerosis with calcification of coronary arteries but no occlusion. There was a bilateral pleural effusion and a terminal bronchopneumonia. The remainder of the organs except as noted below showed no gross lesion.

The findings of chief interest concerned the aorta and the kidneys (Fig. 4). The descending thoracic aorta showed moderate atherosclerosis. The abdominal portion from the level of the cœliac artery down was very grossly involved in severe atherosclerosis with numerous broken down plaques. There were four renal artery orifices all involved in the atherosclerosis. Two on the right side, though somewhat narrowed, were quite patent and led into two large renal arteries. The right kidney was

a little enlarged and weighed 200 gm. On the left side the upper of two renal artery orifices was totally occluded by atheromatous material and the lower one was patent but much reduced, just admitting the stylet of an 18 gauge needle. The left kidney weighed 100 gm. Its upper portion, supplied by the totally plugged vessel, was grossly and uniformly shrunken and dark red in colour. The lower portion retained a more nearly normal appearance.

On section the cortex of the upper pole measured only 0.3 cm. with loss of regular vascular markings in contrast to that of the lower portion which measured 0.8 cm. in width and retained its vascular markings.

Microscopic sections of the left kidney show a clearly defined junction between the better preserved lower pole and the grossly damaged upper pole. In the upper pole there is a marked generalized tubular atrophy and collapse involving both medulla and cortex in consequence of which there is concentration of the glomeruli but distributed in an orderly fashion. Only an occasional glomerulus is sclerosed. On the whole they have retained their architecture remarkably well. In nearly all of them the capillaries contain red blood cells which in many places have conglutinated and appear fused. In the collapsed cortical tubular area the intertubular capillaries are dilated and contain blood. There is a disseminated infiltration with lymphocytes and a relative increase in connective tissue without fibroblastic proliferation. Arteriolar changes are by no means prominent. Slight hyaline swelling is seen in only a few. Some of the small and medium sized arteries show slight hyperplastic sclerosis. In the better preserved lower pole there is a mild degree of hyperplastic arteriosclerosis as in the upper pole. An occasional tubule contains red blood cells while others are plugged with yellowish-brown granules and their epithelial cells are pigmented a similar colour. An occasional convoluted tubule shows granular albuminous degeneration. The capillaries in the glomerulæ and between the tubules are much less prominent here than in the contracted upper pole. A very few hyalinized swollen afferent or efferent arterioles were found.

Sections of the right kidney show no lesion other than a mild degree of hyperplastic sclerosis of a few small arteries and slight hyaline swelling of very few arterioles. A careful search for arteriolar lesions in sections from other organs reveal in the liver, the pancreas and peri-adrenal tissue, a mild degree of hyaline swelling of a few arterioles and a more marked lesion in those of the spleen.

DISCUSSION

Since Goldblatt's classical experiments, it has been definitely established that the hypertension thus brought forth is due to a vasoconstrictor (pressor) substance produced in the ischæmic organ.[2] Furthermore this pressor effect is independent of any nervous origin, since complete denervation or transplantation of the kidney does not abolish the pressor effect. Clinical analogues, with hypertension and occlusion of the main renal arteries, have appeared in the literature.[3] Leiter described a case of chronic hypertension in which necropsy revealed that arteriosclerosis completely occluded one renal artery 1 cm. from the aorta and markedly narrowed the other. Leadbetter and Burkland,[4] and also Boyd and Lewis[5] reported instances in which severe hypertension disappeared following the removal of a kidney which was the seat of arterial narrowing. Homer Smith[6] recently reviewed the literature on hypertension occurring in unilateral urological disease. Dexter[7] reviewed a large volume of literature concerning the search for the theoretical pressor substance, in experimental renal hypertension and in human essential hypertension.

In the cases here described the mechanism of the production of the hypertension in each was undoubtedly the same, but they presented quite different clinical pictures. The explanation probably lies in the dissimilar rate of development of the arterial obstruction. The differential diagnosis is extremely difficult. Case 1 was diagnosed as chronic glomerulonephritis because of the long standing albuminuria and hypertension followed by anæmia and terminal uræmia. Case 2 was diagnosed as malignant hypertension on account of the sudden rise in the blood pressure and the rapidly fatal clinical course. Neurological examination tended to rule out the possibility of a cerebral factor as a cause of the hypertension, while investigation of the suspected renal cause was rendered impossible by the rapid progression of the encephalopathy.

When confronted with a severe unexplained hypertension, obstruction of the blood supply to the kidneys should be seriously considered among the possible causes. The presence of congenital anomalies may be a predisposing factor.

SUMMARY

Gross obstruction at the orifices of the renal arteries, sufficient to reduce materially the renal blood flow, was found at autopsy in two cases of severe hypertension. In both cases the glomerular and arteriolar lesions were not of a degree seen in the kidney of essential hypertension and certainly not proportional to the degree of atrophy and degeneration of the tubules. These tubular changes are, therefore, attributed to the obstruction of the major renal arteries, rather than to the arteriolar or glomerular lesions.

In the first case the presence of nodules of hyperplastic intima, possibly congenital, undoubtedly caused renal arterial obstruction, which was bilateral and equal, thus reproducing in nature the same effect as clamping the renal arteries in the experimental animal. In this case hypertension was followed by renal secretory failure and death in uræmia in three years. The kidneys at autopsy were found to be contracted and granular, weighing only 50 gm. each. The heart weighed 340 gm. and must be considered hypertrophied in proportion to the body weight.

In the second case, with a congenital anomaly, the arterial obstruction was predominantly unilateral and due to arteriosclerosis. The hypertension was less than two years' duration. The development of clinical manifestations was rapid and death occurred before any appearance of cardiac hypertrophy or renal failure. On the right side the orifices of the two renal arteries were moderately stenosed and the kidney weighed 200 gm. But on the left side one of the two renal artery orifices was completely occluded, while the other was markedly stenosed. The kidney was asymmetrically shrunken and weighed 100 gm.

Both cases lend additional support to the theory of a renal mechanism producing human hypertension.

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PATIENTS WITH EMOTIONAL DIFFICULTIES

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THIS discussion is concerned with the very common type of patient in general practice in whom one finds no physical disease, or in whom physical findings are inadequate to explain symptoms. Such patients are at the same time both a frustration and a challenge. In many, the emotional disturbance is obvious, in others, such a factor is strongly suspected or is at least diagnosed in the physician's mind by exclusion. Many are not serious enough to be referred to a psychiatrist and can be satisfactorily handled in a relatively brief time by the general practitioner. In fact, treatment by the family doctor is the only answer to the great problem of the prevalence of emotional disorders seen in private practice. It is for this reason that practical, useful knowledge about these patients is necessary, particularly knowledge of how to talk to them, since treatment is carried out through interview, through "simply talking". The average doctor is often quite skeptical that simply talking with a patient can produce any tangible results. Nevertheless, an abundance of experience proves that this is definitely so. Anyone may prove it for himself in his own practice. The only prerequisites are a sincere desire to help and the full realization that one must treat the patient, not the disease.

DIAGNOSIS

The diagnosis of an emotional disorder must always be a positive one and must never be made simply by exclusion. Thus, if no disorder is found on physical examination, one can say neither that "there is nothing wrong" nor that the patient has a neurosis or mental trouble or "nerves". Of course, there must be something wrong or the patient would never have come in the first place: and the patient is looking to the physician for help with what is troubling him. The positive diagnosis of

emotional disorder can be made on several points: (1) The physical symptoms, which are usually numerous, do not fit any pattern of organic illness, and are in themselves often peculiar and described in odd ways. Thus, the patient complains of "indigestion" caused by one specific food but not by a similar one with the same ingredients. Headaches occur because of some trivial event such as a change in the weather. The symptoms are often described in unusual, metaphorical language. "There seems to be a weight pressing on my head." "It feels like something is clogging my stomach." Some of the commonest complaints are difficulty in sleeping (trouble falling asleep or restless sleep), fatigue (especially in the morning and on slight exertion), "indigestion", tachycardia and palpitation, headache and peculiar feelings in the head, vague complaints referable to the eyes, giddiness and low back pain. The triad of sleeplessness, morning fatigue and numerous vague somatic complaints are strongly presumptive of emotional illness and justify immediate further exploration of the mental area. (2) The presence of mental, as opposed to somatic, complaints. Some of the commonest of these are inability to concentrate, forgetfulness, anxiety in numerous situations, especially when meeting people, and strong feelings of inferiority. (3) The attitude of the patient toward the symptoms. The patient wants to talk about them, seeming in some cases to delight in the discussion. (4) The life history, which when explored, will show previous similar reactions arising in times of stress.

DIFFERENTIAL DIAGNOSIS

Certain mental illnesses may be confused with simple emotional disorders or with psychoneurosis. (1) Early schizophrenia. (2) The depressive stage of manic-depressive psychosis and (3) Organic brain disorders, *e.g.*, brain tumour and G.P.I. The early schizophrenic may have many somatic complaints as described above, but on close observation one will discover apathy or detachment rather than interest and a certain amount of disorganization of thought, ranging from minor irrationality to delusions. The depressive patient usually shows a change in his whole personality and a great reduction of former interests. He will admit spontaneously or on questioning depression, even with suicidal ideas, and often feelings of guilt and unworthiness. The case of brain tumour or G.P.I. may simulate almost any mental condition, but there will usually be some positive neurological or other physical findings.

ESTABLISHING THE DIAGNOSIS

There are two types of cases to be considered. (1) the individual who has been examined with negative physical findings and who is suspected of having psychogenic illness; and (2) the patient who presents on the first interview a picture suggestive of emotional disorder and who has not yet been examined physically. Until one becomes accustomed to diagnosing emotional problems on positive signs, one naturally feels on safer diagnostic ground with the former. However, as soon as there is good reason to suspect an emotional disorder, the physical examination is best deferred (unless there are contra-indications), until one has gone more fully into the history, or even held a few therapeutic sessions. If the condition is of emotional origin, many if not all symptoms may clear up without the necessity of extensive physical examination and expensive laboratory procedures; if not, the organic nature of the symptomatology will become more evident as one proceeds. It is just as bad to miss or neglect an emotional disorder as it is to miss or neglect a physical illness, and still worse to aggravate the emotional disorder by ill-timed examination. In many cases premature or detailed, elaborate investigation serves only to confirm in the patient's mind that there must be something wrong physically and thus make him more difficult to treat. Further, the cost of such investigation may itself produce anxiety.

HOW TO TALK WITH THE PATIENT

Having made a diagnosis of emotional disorder using positive criteria, what is one going to try to do? The goal of treatment is not to do something *for* the patient but if at all possible to help the patient help himself. Every person with an emotional problem, at least theoretically, has within himself the power to get well if only he has help. The doctor's rôle is simply to get him straightened out, to help him understand himself and to mobilize his potential assets, so that he can carry on alone. The difficult thing to remember in treating an emotional disorder is that although the physician says very little and the patient may do

most of the talking, yet the physician is providing truly significant help. It is often not so much what the physician says as his actions and attitudes which bring about recovery.

Helping the patient help himself means that one must help him change some of his attitudes and behaviour. In this way he becomes modified to tolerate situations from which he previously escaped by the development of illness. To do this one must first obtain his confidence and trust and get to know him well. The patient must feel that the physician is sincerely interested in him and is not trying to brush him off. Secondly, the physician must have confidence in and respect for the patient. He must not try to force his ideas upon him nor feel that the patient has to be made uncomfortable before he will "snap out of it". The dignity and human value of the patient must be accepted and the reality of his illness and helplessness understood. These patients are not "just neurotics". An attitude of superiority on the physician's part will prevent effective help.

The first interview in which one attempts to do anything about the patient's problem is very important and a full hour must be allowed. Here, of course, the question of time comes up in the mind of the busy doctor. In many cases the correct approach will in the long run save time. Occasionally the initial one-hour interview is all that is necessary. The conduct of this interview is, therefore, very important. The patient must be allowed to take the lead and to tell the story in any way he wishes. One does not try to get a life history in neat, chronological order. The physician is interested in what the patient has to tell and the way he tells it. Everything the patient says and does is important, so the physician must be a good listener and a good observer. Later the facts can be put together in more orthodox order for confirmation or re-consideration of the diagnosis. If the patient wishes to jump back and forth from his childhood to his present symptoms to complaints about his boss, he should not be interrupted. The interview, of course, can be directed by encouraging conversation about desired subjects. In particular, the patient should be allowed to talk about his complaints. When these are exhausted, the doctor can go on to make a functional inquiry to bring out more complaints if such exist and at the same time, of course, to satisfy himself about the presence or absence of organic disease. At this point one should be careful to obtain all the previous diagnoses that have been given, and the amount and type of investigation. Then the patient may be asked what he thinks is the cause of his illness. Often this question will startle him and will begin him thinking seriously about his symptoms and perhaps about his own personality in a way that he would never do otherwise. Whatever he says, let him talk without criticism or without giving him the feeling that he is boring.

When the patient has completed the description of his symptoms, he will probably have built up the requisite amount of confidence to go on to tell something of his personal problems. If he does not do this of his own accord, he may be stimulated by suggesting that he tell something about himself or simply by saying that one feels there is something more than his symptoms bothering him. This may bring forth a lot of material. In any event the patient should not be unduly probed. Urge him gradually to tell a little more of his life, his work, family, interests, hopes, and ambitions. It is very important here to resist the temptation to jump into explanation or advice, especially if the patient brings out a problem which he has not been able to settle and asks for a solution. The physician's reaction here may be crucial for treatment. If the problem is something that one feels the patient should be able to solve himself, even though not all at once, then it is best not to offer *any* advice. Help him to come to his own decisions by saying "what can you do about" or "let us talk over the possibilities and see if we can not work something out together". Above all, one should avoid offering any advice about physical symptoms of emotional origin.

Inevitably, in talking about himself and his life the patient will show some emotional reaction at some point. The physician must be alert to catch the first signs of this emotion and help the patient to bring it out more strongly. This is best done by approving a show of emotion or by telling the patient you know he must feel angry, sad, resentful, etc. All his emotions must be accepted, even if these include hostility to the doctor himself. This emotional participation is the real key to the patient's recovery; those who do not have it,

but only verbalize, do not have nearly as good a prognosis.

When the hour is over, the patient is asked if he would like to come back for another talk in about a week (very anxious patients should be seen oftener). Some will say they don't think so, at least for a while; many such cases can be considered at least temporarily cured or greatly improved. This is the kind of patient Alvarez, in speaking of functional gastro-intestinal disorders, refers to when he says that many are cured merely by taking enough history. Allowing the patient himself to decide whether he wishes to come back gives him a feeling of independence which will help him to tackle his own problems rather than relying on the doctor to solve them for him. Whether he returns or not, a great deal will go on inside him after he leaves the office, often more than is accomplished in the interview itself. Treatment does not stop but only begins when the patient walks out of the office.

Subsequent interviews do not necessarily have to be an hour long, but it is wise to allow at least half an hour. One will soon see in any individual case how quickly the patient is responding and how much time will be needed.

The next time the patient returns he may say his symptoms are a lot better and begin immediately, with a little encouragement, to talk about his underlying problems. If the symptoms are entirely psychogenic and the interviews are successful, a great many or even all the symptoms will clear up so that an elaborate physical examination is not necessary. Some symptoms, however, may not vanish and the patient may continue to fall back on the belief that there must be something physically the matter. This is the appropriate time for a complete and thorough physical examination. The patient must be told the truth about the findings, even those findings about the interpretation of which the physician is not sure. It does no harm to say that one does not know, it only does harm to have to change one's mind on a dogmatic decision for which there was insufficient evidence. Be definite about negative findings. Do not say there is nothing wrong, but that no physical disease can be found. Often the complete physical examination with frank discussion of the findings will clear up a great deal, if not all, of the problem. This will be so if the patient's main worry has been about some previous medical diagnosis.

When negative findings are offered and discussed, the patient may then ask what is the matter. In this case, it is best to return the question to him to see if he has any idea. It is better for him to say that it is his "nerves" or his worries, than it is for the physician to tell him. Agree with him and ask him if he would like to talk about his worries. If he does not speak of worry, he can be told that physical symptoms can come from such a source. Headaches can be used as an illustration. It will do little good to tell him his symptoms come from worry, because he will probably deny it. On the whole, it is best not to go into detailed physiological explanations of the functioning of organs and how they can be influenced by the mind. This leads the interview away from the patient's underlying problem, and encourages any tendency on his part to verbalize rather than to participate emotionally. The doctor has to watch that he does not try to satisfy his own need to lecture the patient. It is best to treat the whole patient and his problems and let the symptoms take care of themselves.

It is important to remember that there is always some real or imagined stress in the present environment which is contributing to the patient's difficulty and which one can help him overcome. This generally centres on a problem of interpersonal relationships. In fact, the patient will eventually bring out such a problem, and it is by working through present difficulties with him that one best changes his attitudes and reactions. One does not have to feel that because of an unsatisfactory background, a case is hopeless, or that one must go back over the previous life history in detail and interpret it before bringing about a change. The patient must be helped to face and to find solutions for present difficulties if such are possible. Even if there is no solution, the simple fact of having talked over the difficulty with someone who is interested will help the patient to accept it without having to fall back on illness. An emotional re-adjustment is the goal of therapy.

One does not usually uncover the most important problems first. In bringing out his problems the patient at first speaks of the less important ones and gradually moves into the

more important as he gains confidence in the doctor and himself. It has been said that he plays his low cards first. He will usually, however, eventually come around to the thing which is most important to him at that time and, therefore, which is most important to the physician in helping him.

In the process of helping the patient work out his own answers, it may be helpful to suggest to him possibilities of changes in his work and the development of hobbies and recreation. He must never be told dogmatically to do any of these things because then he feels that the doctor is taking the responsibility. He will become less, rather than more, independent and the aim of treatment is defeated. And if desired results are not achieved, of course, he can place the responsibility on the physician.

LENGTH OF TREATMENT

How long does this go on? If the patient is allowed to decide if he wants to come back, it can go on as long as he wants to come. If it is made clear to him that he must co-operate and that his improvement depends as much on himself as on the doctor, and if he is constantly oriented to his present life problems, there will generally not be any great difficulty with lengthy treatments. The patient will tend to wean himself off and will stop coming as soon as he feels satisfied. If, however, he goes deeper and deeper into more problems he should probably be referred to a psychiatrist. If on the other hand, he makes no improvement at all, continually falls back on his physical complaints, or stands helpless in the face of his difficulties, then one is justified in saying, sympathetically, that one cannot help him any more. No great harm is done in stopping treatment in this way. As one gains experience, one soon begins to see what kind of patient can be helped in a short time and what kind cannot.

What is the criterion of cure? The only criterion is the patient himself. When he says he is well, then he is well. If the physician's work is well done, he will not again consult him about physical symptoms of mental origin, but he will always look upon him as a counsellor to whom he can turn in need.

ILLUSTRATIVE INTERVIEW

The following are two condensed, fictitious interviews with a new case, a twenty-two year old single woman, illustrating some of the points discussed in the preceding. Only highlights of the interviews are included.

Good day, Miss Blank. What is troubling you?

I have terrible headaches, doctor.

Tell me something about them.

Well, my head does not really ache—it feels like something is pressing about here on my head (indicates with hand) and I have a tight feeling in the back of my neck. It is pretty hard to describe. Sometimes it is worse than others.

When do your headaches occur?

Oh, they come on any time at all, especially when I am upset.

Have you taken anything for them?

Aspirins—but they do not seem to help.

How do you sleep?

Not very well. I usually lie awake for an hour or two after I go to bed.

Thinking about things?

(Pause) Yes.

How do you feel when you wake up in the morning?

Tired out. I am tired all day long, too. It just seems the last few months the least thing tires me out.

Are you nervous?

Yes, I guess I am. Everything seems to bother me. I didn't use to be like that, but now I get scared at the least little thing. Some days I do not like to go driving, it just makes me so tense.

Do some things bother you more than others?

I get terribly frightened when I am alone in the house or when I have to meet new people.

(Eliciting evidence so far for positive signs of emotional disorder. Now notice change in line of questioning to determine pattern of behaviour to stress.)

Did you ever have anything like this before?

Well, I have always been a nervous person. In school I used to get so scared around examination time that I used to get just sick.

Did you ever have a nervous breakdown?

Well, I suppose you would call it that. I had to be off work once for a couple of months because I just could not stand the strain any longer.

(Next, shift of emphasis.)

Perhaps you are worried about something now?

(Pause) Well, I am worried about my job a bit.

(Patient playing a low card.)

How is that?

I get upset and nervous when I have something to do that is a bit out of the ordinary. It just seems too much for me. That's when I get my worst headaches.

Do you think there is a connection between your headaches and getting upset?

(Testing patient for possibility of acquiring insight.)

Yes, I think that is what causes them most of the time, you know.

Maybe you are worried about some thing at home.

(Pause) Well, I do not think so. I get along pretty well with my mother but sometimes she upsets me.

How is that?

(With growing feeling) Oh, she gets me so irritated. She is always trying to tell me what to do and always asking me where I have been and how I spent my time and what I am going to do tomorrow and who I was with and things like that.

You get a little mad at her sometimes?

(Encouraging expression of emotion, especially negative feelings.)

Well, no—yes, maybe I do. I would like to tell her off sometimes.

You do not like to be ordered around. You like to be independent.

(Bringing out the patient's strength.)

Yes, I certainly do, and she will not let me, that is what makes me so mad. What can I do about it?

What do *you* think would be the best thing to do about it, Susan?

Well, I suppose I could stand up for myself a little more. I might just go ahead and do some things I want to and not even tell her about them when she starts to ask questions.

That might lead to a quarrel.

(Helping patient face the possible results of the new behaviour she must adopt.)

Yes, it might. (Pause) Well, I do not care. I have put up with it for a long time.

I see our time is about up. Would you like to come back again for another talk?

Yes, I would. I think this has helped me a lot.

SECOND INTERVIEW

How are you today?

A lot better, doctor. I had quite a fight with my mother. Well, it was not really a fight. I wanted to go to a party and she said I needed to stay home and rest. I just decided to go anyway. She made quite a fuss, but in the end I went.

Did you have a good time?

Yes, I did and I felt a lot better the next day, did not feel tired like she said I would. Mother tried to ask me a lot of questions about it, but I just said I had a good time and I was old enough to look after myself.

How are your headaches?

I have not had so much trouble this last week. In fact, I really have not had a bad headache since I was here.

Do you think there is any connection between your headaches and your difficulties at home?

I think that is the main connection. I have noticed that about the only time I do get a headache is when I have had some difference with my mother, or feel mad at her.

(Patient developing insight. Note how she is led to this, and that the symptom is not discussed in isolation.)

Now you are standing on your own feet more, it isn't so bad, is it?

No. I seem to be sleeping better, too.

I'd like to give you a physical examination just to be sure we are not missing anything.

(After physical examination, which is negative.) No physical disease—perfect physical condition. Do you think you can handle yourself now or would you like to come back for another talk?

I do not think I will come back for awhile. I think I see what a lot of the difficulty is and I will have to see what I can do about it myself.

Is there anything you would like to ask me before you go?

Can I come back in a few weeks to tell you how I make out?

Certainly. Good luck.

RÉSUMÉ

Il n'est pas de jours que le praticien ne recontre en clientèle plusieurs patients chez qui il ne peut rien relever d'anormal ou dont les plaintes ne s'expliquent pas objectivement. Certes un psychiâtre est nécessaire dans les cas de troubles émotifs graves, mais c'est en somme au praticien d'user de sa remarquable influence pour résoudre ces petits problèmes journaliers, ennuyeux certes pour lui, mais importants pour le patient. Il se doit donc d'avoir sur ce sujet des connaissances pratiques, notamment pour ce qui regarde la façon de parler à son patient et l'inciter à se confier pleinement en lui. Ici plus qu'ailleurs triomphe l'aphorisme: il n'y a pas de maladies, il n'y a que des malades.

Le diagnostic d'un trouble émotif doit être un diagnostic positif, il ne suffit pas d'y arriver en procédant par élimination. L'examen physique est-il négatif, on n'a pas le droit de dire au patient qu'il n'a rien ou qu'il ne s'agit que de "ses nerfs".

C'est un malade qu'il faut traiter, pas nécessairement en faisant quelque chose pour lui, mais plutôt en l'aidant à s'aider lui-même. Il possède en lui-même le secret de sa propre guérison, le pouvoir de finir par tolérer des situations plus ou moins pénibles dont il s'échappait auparavant en développant des symptômes physiques de maladie. S'il sent que le médecin s'intéresse à son cas, patiemment et sans signes d'humeur, il lui racontera sans contrainte tous ses troubles; et c'est au cours de cet entrevue qu'une réaction émotive à un même sujet donné mettra le médecin sur la piste du facteur déterminant. Il lui suffira alors de rassurer le patient sur ce point et lui donner les directives qui conviennent. Quel est le critère de guérison? C'est le patient lui-même. Quand il avouera se sentir mieux, c'est que vraiment il sera mieux.

BARBITURATE POISONING TREATED WITH MODIFIED ELECTROTHERAPY

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BARBITURATE poisoning is increasing in frequency. Emergency treatment with such preparations as metrazol, picrotoxin and autonomic nervous system stimulants such as benzedrine sulphate, are not very successful because of their transient action and in some cases because of the toxic action of the drug itself. It is also believed that pulmonary infection is a common sequela. Furthermore, epileptiform seizures are liable to follow the administration of metrazol and picrotoxin, thus complicating the patient's condition.

Some psychiatrists know from experience with electro-stimulation of the brain that certain electro-stimuli appear to aid in the elimination of barbiturates within a very short period of time, *e.g.*, 14 grains of sodium pentothal administered intravenously within two minutes. This observation suggested the use of selected electro-stimulation in the treatment of patients suffering from over-dosage with barbiturates. The following case illustrates a new method of treating barbiturate poisoning. As far as is known this method has not been reported in the literature.

Mrs. A.B., aged 49, married, no children. The patient has always been delicate. Her husband has a pension for silicosis and this couple live on his pension of $90.00 per month, and from the material point of view this patient has little to live for.

The patient was examined two years ago for a complaint indicative of menopausal changes. Appropriate treatment gave symptomatic relief. Five months ago she presented herself for further examination for a hæmorrhagic vaginal discharge. A gynæcological examination suggested a diagnosis of degenerating carcinoma of the cervix. The patient herself believed that she was suffering from cancer and was showing evidences of depression. She had a total hysterectomy, but pathological diagnosis did not reveal any evidences of malignancy. Convalesence appeared normal, but the patient left the general hospital in a depressed condition.

About the end of Setpember, 1950, she was again requiring medical attention and it was noted at this time by one of us (C.P.) that she was depressed, irritable, weeping at frequent intervals, complaining of hot flushes, eating and sleeping poorly. The patient was thin and emaciated. Œstrogenic substances were prescribed and the patient was given a prescription of 36 x 1½ gr. capsules of pentobarbital sodium with instructions how to take them.

On October 2, the husband found his wife lying on the floor of his home at approximately 11 a.m. She told her husband she had taken "too many sleeping pills by mistake". Within a short time the patient was unconscious and was removed to a general hospital. On

arrival she was stuporous, flushed, reflexes present, breathing normal and rousable by painful stimuli. As more than 1½ hours had elapsed since the patient had taken the sleeping pills, gastric lavage was not done. At this time no knowledge as to the quantity of sedation taken was available.

The patient received such preparations as coramine and methedrine and was placed in the oxygen tent. Her condition remained satisfactory with her blood pressure averaging 104/72 until 1.30 p.m., when she became deeply comatose with shallow respiration and imperceptible pulse. All reflexes, including corneal reflexes, disappeared.

The patient was seen by one of us (J.J.G.) at 2 p.m. She was in deep coma; her pupils were contracted and did not respond to light; corneal reflexes absent; deep reflex absent; positive Babinski on the right side; breathing shallow; pulse imperceptible; blood pressure 88/?.

Brain stimulation was commenced at 2.24 p.m. The electrodes were placed immediately above the ears and a modulated electric stimulus was applied. Immediately the cardio-respiratory picture improved. Pulse became full and strong at 88 per minute and remained at this all during the treatment. The blood pressure rose to 130/76 immediately, breathing became strong and regular and the patient's general appearance became more satisfactory. There was little in the way of muscular twitching and there was little pyramidal response during the first three hours of treatment. The electric stimulus flowed continuously until approximately 5 p.m., the electrodes being in the same position all during this time. It was noticed at this time that the patient's condition was gradually worsening and the treatment was interrupted for a few minutes to enable her to receive the last rites of her church. At this time blood pressure had dropped below 80 and the original picture had re-established itself. Electro-stimulation was commenced again, but this time the electrodes were moved from place to place as it appeared evident that the patient had developed a tolerance to stimulation over the parietal regions and was not responding. Her condition again showed dramatic improvement with the changed positions of the electrodes. They were constantly moved from one position to another, ranging from the bi-temporal position all the way down to the ['es of the jaw. Motor response reappeared at 5.15 . and at 9.30 p.m. tendon reflexes returned in rapid succession. At 10 p.m. the patient made voluntary movements of the hands, arms and legs and was able to swallow sips of water. At 10.30 p.m. she could be roused by oral stimulation. At 11.30 p.m. she was fully conscious and able to answer questions without confusion. The electric current had been flowing almost continuously from 2.24 p.m. until 11.30 p.m. The patient required some further medication during the night, but no relapse occurred. Examination of the patient the following day revealed that despite such prolonged brain stimulation with electricity no confusion or memory loss were noted. It was later learned that the patient had taken 29 1½ gr. capsules of pentobarbital sodium and that she had planned suicide for some weeks.

The patient's depression was not helped by this type of electric stimulation. She attended Homewood Sanitarium as an out-patient for electro-convulsive treatment and after two such treatments it was noted that her depression and ideas of hopelessness had disappeared. She was allowed home from the general hospital and attended Homewood as an out-patient for a further two electro-convulsive treatments. Her mental condition was very satisfactory after her fourth treatment and it was noted that she had gained weight, was relishing her food, was sleeping well and was definitely hopeful as to the future.

This case is remarkable for the following reasons:

1. The large dose of barbiturate consumed.

2. The lack of an important first aid measure, namely, the gastric lavage.

3. The prolonged and succesful use of brain electro-stimulation.

4. The patient's rapid recovery following electro-convulsive treatment.

The apparatus used is known as the Reiter Electro-Stimulator, Model No. CW 47. As to the mode of action of this treatment the following opinions are suggested tentatively:

1. Cardio-respiratory stimulation (electrical artificial respiration) carries the patient along until the drug is in some way neutralized or eliminated.

2. Electric brain stimulation in some way causes the rapid elimination of barbiturate drugs from the nervous system.

3. This type of brain electro-stimulation cannot be used therapeutically without considerable pre-treatment sedation, *e.g.*, five to ten grains of sodium pentothal intravenously, on account of severe pain. Painful stimulation of such an intense nature may be a factor in facilitating the return of consciousness. This patient did not complain of any pain during her treatment, although it was obvious from her response during the latter part of the treatment that consciousness to such painful stimulation was returning.

It is believed by us that this form of treatment might be of great value in the treatment of coma due to morphine, alcohol or insulin (the so-called irreversible coma.)

RÉSUMÉ

Les auteurs rapportent le cas d'une femme qui s'empoisonna accidentellement avec une forte dose de barbiturates, et qui reprit assez rapidement conscience grâce à une méthode nouvelle de traitement consistant en une stimulation électrique du cerveau. A cause de la fréquence de plus en plus grande des empoisonnements par les barbiturates, cette méthode, dont il n'a jamais été question dans la littérature médicale, semble s'avérer fort efficace, beaucoup plus que les médications d'urgence usuelles telles que la picrotoxine, le metrazol ou la benzidrine, dont les effets sont transitoires et non dénués de toxicité. Ce cas fut remarquable à cause de la forte dose de poison qui fut ingérée, l'absence de lavage gastrique comme mesure d'urgence, et la ranimation rapide et complète de la malade à la suite d'un traitement prolongé.

Quant au mode d'action de cette électro-stimulation, les auteurs sont d'avis que ces secousses électriques au cerveau provoquent par un mécanisme quelconque l'élimination ou la neutralisation des barbiturates du système nerveux. La douleur qu'elle cause peut n'être pas non plus étrangère au regain de la connaissance. A cause de cette réaction douloureuse au traitement, il est de bonne pratique de le faire précéder d'une médication sédative énergique, par exemple, l'injection intraveineuse de cinq à dix grains de sodium pentothal. Les auteurs croient que cette thérapie peut s'avérer très utile dans le traitement du coma provoqué par la morphine, l'alcool on l'insuline.

THE USE OF TRICHLORETHYLENE IN OBSTETRICAL ANALGESIA AND ANÆSTHESIA

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DURING recent years the use of trichlorethylene in obstetrics has increased, and is becoming more and more popular. From August 1949 to October 1950 we collected a series of 900 cases in which trichlorethylene was used (*a*) as an analgesic, and (*b*) as an anæsthetic agent in obstetrics. The observations on these cases are the basis of this paper.

The agent used in this series was "trilene" which is a specially purified form of trichlorethylene manufactured by Imperial Chemical (Pharmaceuticals) Ltd., Manchester, England.[1] The chemical and pharmacological properties of this drug have been described in many standard textbooks and journals.[2, 3, 4, 5]

ANALGESIA

Procedure (using Cyprane inhaler[1]).

The inhaler was given to primiparæ when the cervix was 7 cm. dilated and to multiparæ when the cervix was 5 cm. dilated, and the patients had received analgesic drugs such as: morphine, heroin, hyoscine, demerol and/or seconal earlier in labour. Once the Cyprane inhaler was put into use these analgesic drugs were discontinued.

The mixture-adjusting collar on the inhaler was locked at the fourth line and the inhaler was filled with 15 c.c. of trilene. The patient was instructed to apply the mask to her face and take two or three deep breaths when she felt the beginning of a pain. She was also told to remove the mask when the contraction had ceased. The inhaler was then attached to the wrist of the patient by means of a bracelet to prevent it falling to the floor. The anæsthetist remained with the patient until he was satisfied that she was using the equipment properly. The patient was told to notify the nurse when she could no longer detect the odour of trilene as this would indicate that the supply of the agent had been exhausted. Before refilling, the trilene container was checked to make sure that it was absolutely empty. This precaution was taken to prevent overfilling of the inhaler which might result in burns of the face. The patient continued to inhale the agent until ready for delivery.

In the following chart 10 cases are presented. These were selected at random from our series of 100.

Observations.—(1) The patients, generally, were pleased with this form of analgesia. (2) Labour did not appear to be appreciably lengthened. (3) 4% of patients were uncooperative. (4) 1% of patients were nauseated. (5) 3 cases where the baby required resuscitation could not be attributed to trilene. (6) The patients used contractions to better advantage. (7) It was estimated that 15 c.c. of trilene gave approximately 1½ hours of analgesia. However, there was great variation because of differences in patients such as pain threshold, frequency of pains, rate and depth of respiration. The quantity of trilene was also dependent upon the temperature of the inhaler.

ANÆSTHESIA

A. *Trilene and air* (Cyprane inhaler).

A total of 510 cases were delivered under trilene and air using the Cyprane inhaler. This form of anæsthesia was administered both by the anæsthetists and obstetrical interns. The following procedures were carried out: spontaneous delivery, episiotomy and repair, forceps extraction, breech delivery, rotations, manual removal of placenta, and twin deliveries.

Technique.—All patients received atropine gr. 1/150 for pre-anæsthetic medication. The face-mask was applied with the mixture-adjusting collar set at the first line and the concentration was increased as tolerated by the patient until "full on". After the patient had been anæsthetized, which usually took 5 minutes, the patient was ready for delivery.

Observations.—(1) The baby's breathing was not depressed as much as with other inhalation agents. (2) Vomiting was very rare. (3) Cardiac arrhythmias were not detected. (4) Tachypnœa decreased as experience in administration was gained. (5) No cases of respiratory arrest occurred. (6) Muscle tone was not decreased. (7) Anæsthesia and amnesia were excellent. (8) Rapid induction and recovery. (9) Post-anæsthetic comments regarding the anæsthetic by the patients were generally favourable to the agent used. (10) Average quantity of trilene used per delivery was 30 c.c.

TABLE I.

OBSERVATIONS ON THE SELF-ADMINISTRATON OF TRILENE FOR ANALGESIA DURING LABOUR

Age and parity	*Fetal position*	*Level presenting part*	*Dilation cervix cm.*	*Frequency of pains (minutes)*	*Obstetrical complications*	*Setting of mixture-adjusting collar*	*Trilene used c.c.*	*Trilene adm. hours*	*Comments on reaction and appearance of patient under analgesia*	*Anæsthesia for delivery*	*Minutes of anæsthesia for birth*	*Condition of baby at birth*	*Operative procedure*	*Anæsthetic complications*	*Comments of patient after complete recovery*
20 I	R.O.T.	S.P. −1	7	2–3	Nil	4	30	3	Patient ceased screaming and pain seemed to be relieved	Trilene N_2O	10	Cried spontaneously	Episiotomy repair	Nil	"I only remembered when membranes ruptured; don't remember pain."
24 I	R.O.A.	S.P. −1	Full	1–2	Nil	4	10	2½	Pains were relieved and patient was lying quietly.	Trilene air	10	Cried spontaneously	Low forceps; episiotomy repair	Moist during anæsthesia	"Remember my labour but trilene did help."
31 IV	L.O.T.	S.P. +1	8	1–2	Nil	4	25	1½	Appears to be getting relief; doesn't speak English or French.	Trilene air	2	Cord twice around neck; required resuscitation	Spontaneous	Nil	"Trilene helped pains and I liked it." (per interpreter.)
34 II	L.O.A.	S.P.	6	2	Nil	4	30	5¼	Patient very cooperative; resting peacefully. Almost complete relief of pain.	Pontocaine spinal	35	Cried spontaneously	Low forceps; episiotomy repair	Nil	"Wonderful. Made me very sleepy."
33 II	R.S.P.	S.P. +1	8	2	Breech	4	15	3½	Asked for inhaler when it was taken away.	Trilene air	7	Cried spontaneously	Episiotomy; Mauriceau-Smellie-Viet manœuvre	Nil	"Trilene was very pleasant and it helped me."
28 III	L.O.A.	S.P. +1	7	2	3½ wks. premature; spotting	4	5	½	Patient very cooperative; appeared to blunt the pain.	Pontocaine spinal	15	Cried spontaneously	Low forceps; episiotomy repair	Nil	"It helped me but made me feel groggy."
20 III	R.O.A.	S.P. +1	6	2	Nil	4	5	½	Patient noisy and restless after using inhaler.	Trilene N_2O	7	Cried spontaneously	Spontaneous delivery	Nil	"I remember everything that happened but did not feel pain."
33 IV	L.O.A.	S.P. +2	8	3	Nil	4	10	¾	Eased the pain; patient slept between pains.	Trilene air	5	Cried spontaneously	Spontaneous delivery	Nil	"A wonderful way to have a baby. No pain; pleasant dreams."
31 I	L.O.P.	S.P. +2	8	2	Posterior contracted pelvis	4	15	2½	Patient relaxed and her pains were blunted.	Pontocaine spinal	15	Required prolonged resuscitation	Mid forceps; episiotomy repair	Nil	"Trilene was very good; the pains were not as severe."
18 II	R.O.A.	S.P.	6	2	Nil	4	12	½	Very co-operative; pains blunted; patient relaxed.	Trilene air	8	Cried spontaneously	Spontaneous delivery	Nil	"A definite relief of pain. Preferred to spinal which I had during labour for my first baby."

B. *Trilene and nitrous oxide and oxygen.*

A total of 390 cases were delivered using trilene in combination with nitrous oxide and oxygen. This technique was used mainly by anæsthetists and was their method of choice. All of the procedures performed under trilene and air were done with this technique also.

Technique.—To the outflow side of the standard Heidbrink Kinetometer was attached either a McGill inhaler[6] or a Foregger vinethene vaporizer connected by a rubber tube to a 3 litre bag which was attached to a Foregger face mask with exhalation valve left open.

Nitrous oxide and oxygen were run through at the rate of 5 litres of each per minute. This was in order to keep the bag full and to prevent the accumulation of carbon dioxide.[7] The face mask was strapped in place and the nitrous oxide and oxygen mixture started, following which the trilene was cut into the system. The concentration was gradually increased until the patient was anæsthetized. Thereafter the trilene was gradually reduced as the operation proceeded. The maximum dial setting was rarely more than "half on". Induction was usually completed in 3 minutes.

Observations.—(1) Induction by this method was more rapid and smoother than with the technique described previously in this paper. (2) Greater relaxation was obtained when using nitrous oxide and oxygen with the trilene than that obtained with air. (3) The infant's respirations were not noticeably depressed. (4) Vomiting was rare but more frequent than with trilene-air mixture. (5) No post-anæsthetic sequelæ occurred. (6) Average quantity of trilene used per delivery was 10 to 15 c.c. (7) One patient had a prolonged period of emergence. (8) One patient salivated profusely because the administration of the atropine was badly timed. (9) A mother with myasthenia gravis was delivered of a baby also suffering from this disease without any untoward effects to either. (10) Respirations were slower than with the trilene-air technique.

CONCLUSIONS

In our estimation, trichlorethylene is an excellent agent for analgesia and anæsthesia in obstetrics because: (*a*) it is relatively safe and efficient; (*b*) very few side effects occur; (*c*) it is received with enthusiasm by the patients; (*d*) it is comparatively inexpensive because only a small quantity is used for each case.

We wish to thank Dr. G. A. Simpson of the Royal Victoria—Montreal Maternity Hospital for his co-operation in supplying clinical facilities for this series of cases and we also wish to thank Dr. F. A. H. Wilkinson of the Royal Victoria Hospital for his constructive criticism in the preparation of this paper.

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RELATIONSHIP OF FIBRINOGEN B TO THROMBOEMBOLIC PHENOMENA

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IN a paper by Cummine and Lyons[1] in 1948 it was claimed that, with the aid of a test for a newly discovered substance in the blood which they called fibrinogen B,[2] those persons, in particular postoperative patients, could be selected in whom thromboembolic phenomena were going to occur some 12 to 24 hours prior to the onset of the clinical manifestations of phlebitis or pulmonary embolism. If this could be done the problem of pulmonary embolism would be practically solved, for in the anticoagulants we have potent agents for preventing or controlling intravascular clotting[3] if we are alerted in time. These authors felt that in the vast majority of cases, if not invariably, it was necessary to have fibrinogen B in the blood before thrombosis could occur. They also claimed that there was a readily demonstrable "prethrombotic state" in which the blood-clotting time is depressed to the region of three minutes for a period of several hours, usually some 12 to 24 hours prior to the onset of clinical thrombosis. It was their contention that if a prethrombotic state were proved and there was fibrinogen B in the blood, the occurrence of thromboembolic phenomena was inevitable.

METHODS

A number of fibrinogen B tests and clotting times were carried out during a period from October 1, 1948, to December 15, 1948, on normal and pre- and post-operative patients on Ward C of the Toronto General Hospital. Some tests were also carried out on obstetrical patients in the Burnside Division of the Toronto General Hospital between March 10, 1949, and the end of May, 1949.

The test for fibrinogen B which we used was a later modification by Lyons[4] of the one described in his original article.[1] This modified test gave much more consistent results than the previous one and got around the variability due to the different sized drops of reagent which might have been used. It was carried out as follows: To a 15 c.c. centrifuge tube marked at the 5 c.c. level 0.5 c.c. of 1% sodium oxalate solution was added and then the blood to be tested was added up to the 5 c.c. mark. The solution was thoroughly mixed and the tubes centrifuged to obtain the plasma; 2 c.c. of plasma were pipetted off and 1 c.c. placed in each of two small test tubes. Using a syringe with a 20 gauge needle as a standard dropper, to one of these tubes drops of the fibrinogen B reagent[1] were added while shaking gently until the solution became definitely opalescent. To the other tube 1 drop less of the fibrogen B reagent was added so that a fainter opalescence was obtained. Usually from 10 to 14 drops were required in the first tube. The tubes were then set aside and left undisturbed for ten minutes. Readings were then made on the second set of tubes by tilting them and viewing them by transmitted light. In the negative tests the solution was mildly milky but essentially homogeneous and flowed easily. In what we considered as doubtful or suggestive cases there was some whitish fibrinous precipitate but no definite gel formation. These cases should likely be considered as negative. In the positive cases there was a definite gelling tendency such as described in the original article.[1]

Blood clotting tests were carried out at intervals on patients showing fibrinogen B in their blood using a freely moveable glass bead in a small glass tube. The clotting time was taken as the time between the removal of the blood from the vein until the bead ceased to move on inverting the glass tube. No attempt was made to follow the clotting time over several hours in those persons with clotting times in the region of three minutes, as advised by Cummine and Lyons, to clearly demonstrate the prethrombotic state, as this was impractical under our working conditions and also as we were chiefly interested in studying fibrinogen B itself. A definite attempt was made to follow patients who were undergoing operation both pre- and post-operatively. Tests, in general, were made every second or third day postoperatively over a period of about ten to twenty days.

RESULTS

Three hundred and forty-four fibrinogen B tests were carried out on 172 persons on Ward C at the Toronto General Hospital. Twenty-six normal persons (healthy, working individuals) were tested, and 24 tests were negative and 2 were doubtful. It was, therefore, felt that it could be concluded that fibrinogen B was in general absent from the blood of normal individuals. Thirty-three patients were followed well postoperatively, having at least 2 fibrinogen B tests postoperatively with an average of 3.85 tests per patient. Of these 29 showed fibrinogen B in their blood. The 4 patients that did not had had the following operations; one each of a Smith-Peterson hip nailing, an interval appendectomy, a herniorrhaphy and a thyroidectomy. Thus 88% of patients having operations on a general surgical ward showed positive fibrinogen B in their blood.

In this group of 33 patients there were 4 who showed some fibrinogen B in the blood preoperatively. These were 4 women, one with an œsophageal hiatus hernia and gall stones, one with a 40 lb. ovarian cyst, one with cancer of the breast, and an Indian with a thyroid adenoma.

Considering the postoperative results according to the types of operations it was found that all cases of gastrectomies, mastectomies, cholecystectomies and splenectomies showed fibrinogen B in the blood in the postoperative period. Most appendectomies, Smith-Peterson nailing of hip and extraperitoneal lumbar sympathectomies on the other hand did not show fibrinogen B in the blood postoperatively. In general, therefore, the more major the surgical procedure the greater the likelihood of fibrinogen B being demonstrable in the blood in the postoperative period.

Of the total cases studied 8 showed fibrinogen B in the blood in association with a clotting time of three minutes or less. From the work of Cummine and Lyons we would expect that these persons would be the ones most liable to develop phlebitis, but would not feel that thrombosis was inevitable in them, as the depression of the clotting time to the three minute level was not proved over a period of several hours as they feel is necessary for the establishment of the "prethrombotic state". Of these 8 cases 2 showed mild signs of phlebitis. There were no clinical signs of phlebitis or pulmonary embolism in the other patients studied in whom the clotting times were above the three minute level. This suggests that there may be a demonstrable prognostic prethrombotic state such as claimed by Cummine and Lyons. Further work on this problem would be of value.

As to the time relationships for finding fibrinogen B in the blood postoperatively, it was found that, grouping the second and third postoperative days in these cases 73% of 15 cases showed a positive test for fibrinogen B. Grouping the 7th, 8th and 9th postoperative days, 84% of 25 cases gave a positive test. On the 12th, 13th and 14th days 66% of 15 cases gave a positive test, and between the 15th and 23rd postoperative days 33% of 15 cases gave positive tests. It thus appears that postoperatively a large number of cases (roughly 70%) show fibrinogen B in the blood by the second day but that the percentage of cases showing fibrinogen B in the blood further increases to a maximum of about 85% around the 8th postoperative day, and then decreases to around 66% by the 13th day, and

to 33% positives in the 3rd postoperative week. According to Cummine and Lyons[1] a decrease in fibrinogen B in the blood can be due either to a clearing-up of the factors leading to fibrinogen B formation in the body, or to its utilization in the formation of intravascular clots. As no evidence of phlebitis was found in the majority of these cases (97%), it would seem that in general, between the 14th and 21st postoperative days the factors leading to fibrinogen B formation must cease to be operative. This fits in with the clinical finding that the incidence of thromboembolic phenomena falls off greatly after 14 days have elapsed from the time of operation. The fact that the occurrence of fibrinogen B in the blood postoperatively tends to be highest around the 8th day also fits in with clinical findings in that this is about the most dangerous time for the initiation of thromboembolic phenomena.[5]

As well as in the postoperative group positive fibrinogen B tests were found in 35 other persons having miscellaneous conditions. These included 4 fractures, 4 inoperable carcinomas of the large bowel, 3 bleeding duodenal ulcers, 4 large abscesses, 2 cases of extensive bruising, 2 cases of Buerger's disease with gangrene, and 2 cases of varicose veins with ulcers. These in general conform to Cummine and Lyons' view that collections of necrotic tissue, blood, or pus, predispose to fibrinogen B formation, and hence possibly to intravascular clotting.

What is the effect of heparin and dicoumarol therapy on the fibrinogen B in the blood? Lyons[4] stated that heparin did not prevent its formation while dicoumarol usually tended to do so, and our experience would confirm this, for 3 persons on heparin continued to show positive fibrinogen B tests while 5 persons on dicoumarol did not.

In our investigation of obstetrical patients some 130 tests were carried out on 78 persons. It was found that of 47 normal women in labour 22 were positive for fibrinogen B and 23 were negative, with two cases in the doubtful category. Apparently 50% of normal women in labour show fibrinogen B in their blood. It would seem likely that the fibrinogen B positive individuals would be the ones most likely to develop post-partum phlebitis.

Five pre-eclamptic patients near term all showed fibrinogen B in the blood. It would thus seem that a toxæmia of pregnancy would predispose to phlebitis.

DISCUSSION

Our studies would indicate that fibrinogen B is a distinct substance which is readily demonstrable by a relatively simple laboratory test. The fact that blood containing fibrinogen B readily gives a gel formation in contrast to normal blood which does not, would make it seem logical to assume that its presence in the blood would predispose to intravascular clotting such as phlebitis. On the other hand its presence alone, as we have amply demonstrated in our tests, does not lead to thromboembolic phenomena. The fact, however, that no cases of phlebitis were recognized in persons not showing fibrinogen B in the blood suggests that its presence may be a necessary prerequisite for intravascular clotting to occur.

The fact that, as we have found, fibrinogen B is present in the blood of practically all patients following major operations would suggest that under these circumstances the test would not need to be carried out routinely but that its presence in the blood could be assumed. Thus while fibrinogen B may be an intermediate in the clotting mechanism whose presence in the blood is necessary for thrombosis to occur, still its almost universal presence in the blood after any major operation means that the demonstration of its presence, under these circumstances, is of no practical value in predicting those in whom thromboembolic phenomena may occur. Some other test must be devised for this purpose and it may possibly be that repeated clotting time determinations with the demonstration of a prethrombotic state as suggested by Cummine and Lyons may be the answer.

The fact that necrotic tissue predisposes to fibrinogen B formation would emphasize still further the recognized importance of atraumatic surgery, with the minimization of tissue damage and the avoidance of mass ligation of tissues. Rough surgery would definitely seem to predispose to thromboembolic phenomena.

As to why the incidence of occurrence of fibrinogen B in the blood should reach a maximum in the period of from 7 to 9 days postoperatively no definite answer can be given. It may, however, be that revascularization of the necrotic tissue must occur in order to get enough absorption to lead to the maximum formation of fibrinogen B in the blood.

The fact that in our experience fibrinogen B

tended to be most commonly present in the blood between the 7th to 9th postoperative days would suggest that this is likely the most dangerous period for thrombosis to occur. This fits in with clinical experiences in that thromboembolic phenomena usually do not occur before the second postoperative week.[5] Taken together these facts would suggest that if anticoagulant therapy were, in the presence of any suspicious clinical signs, initiated six or seven days postoperatively, and carried on for a week or ten days, thromboembolic phenomena would be prevented or aborted, and yet the danger of producing hæmorrhage at the operative site would be almost infinitesimal.

While it is recognized that in some instances the number of cases involved is small it is our opinion that the following conclusions can validly be drawn.

CONVULSIONS

1. Fibrinogen B is a substance not found in the blood of normal individuals, but under certain abnormal physiological conditions can readily be demonstrated in the blood by a simple laboratory test.

2. The mere presence of fibrinogen B in the blood is in no way prognostic of the occurrence of thromboembolic phenomena. As the majority of postoperative patients (88%) show fibrinogen B in their blood its demonstration, under these circumstances, is of no practical value in selecting the ones in whom thrombosis will subsequently develop.

3. Tissue necrosis of any great extent with or without added infection leads to the presence of fibrogen B in the blood.

4. Heparin does not interfere with the formation of fibrinogen B while dicoumarol may do so.

5. Fifty per cent of normal women in labour show fibrinogen B in the blood and 100% of pre-eclamptic patients near term do so.

6. There is some evidence to suggest that the maximum postoperative incidence of fibrogen B in the blood may occur approximately one week after operation.

I wish to express my appreciation to Dr. Gordon Murray for his interest and encouragement in this work; to Dr. Joselyn Rogers for his co-operation in obtaining blood specimens from obstetrical patients; and to the interns and medical students on Ward "C", Toronto General Hospital, who assisted in obtaining the blood specimens for this work.

This work was done while resident on Division III General Surgery (Wd. C) at the Toronto General Hospital under the direction of Dr. Gordon Murray.

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PRIMARY CARCINOMA OF THE LIVER*

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PRIMARY carcinoma of the liver is a relatively rare disease and has always been considered a pathological curiosity of no surgical importance. The diagnosis is seldom made clinically in the early stages, and until recently the technical difficulties of liver resection have been so great that successful removal of such tumours has rarely been accomplished.

In the past few years with advances in the treatment of shock and infection and the development of new methods of hæmostasis, removal of part of the liver has become feasible. Many cases of survival following partial hepatectomy have been reported. At present, benign tumours offer the best prognosis but with earlier diagnosis some cases of primary carcinoma of the liver may be amenable to surgical treatment. It therefore seems worthwhile to review the proved cases of hepatoma in order to clarify the pathological and clinical features.

Incidence.—Most reports in the literature place the incidence of primary liver carcinoma between 0.1 and 0.3% of routine autopsies. These figures do not give a true incidence because many cases proved by laparotomy and biopsy do not die in hospital.

At the Royal Victoria Hospital there have been 24 cases of primary carcinoma of the liver in the past 25 years. Of these, 9 have come to autopsy; in the remaining 15 the diagnosis was established at operation and confirmed by biopsy.

Carcinoma of the liver has been reported in all age groups but the majority of cases occur

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in the fifth and sixth decade. In this series the youngest case was 22, the oldest 69 and the average age was 50 years. Seventeen cases occurred in males, seven in females. This increased prevalence in males has been noted in all previous reports and several theories have been advanced in explanation. The frequent association of carcinoma with cirrhosis of the liver has been mentioned as a determining factor. In five of our cases cirrhosis was present. This is a lower incidence of association than has usually been noted but it is rather difficult to draw conclusions on the basis of such a small series.

Clinical features.—The diagnosis of primary carcinoma of the liver is rarely made before operation or autopsy. In reviewing this series it becomes apparent that these cases do follow a similar pattern. The most common initial complaint was abdominal pain, which was present in twenty cases. This was usually a constant dull aching sensation in the upper abdomen, but occasionally the patient complained of intermittent attacks of severe colicky pain, probably due to hæmorrhage within the tumour. Anorexia and weight loss were noted in 17 patients and in 4 vomiting was a prominent feature. Jaundice was present as an initial complaint in only two cases, but developed commonly as a terminal event associated with cachexia and ascites. In 9 cases the earliest finding was an abdominal mass or swelling. The duration of symptoms was short, ranging from one week to one year with an average of seventeen weeks.

Examination revealed no characteristic findings except for a mass in the upper abdomen which was palpable in half the cases.

Laboratory findings gave little aid in diagnosis. Anæmia was universally present, usually of a hypochromic microcytic type. Liver function tests showed impairment in only five patients and these were the cases in which cirrhosis was present.

Radiological study suggested the diagnosis in several patients because of a soft tissue mass or pressure effects on neighbouring organs. In three instances the right diaphragm was elevated, in one the transverse colon was displaced downward and in two an extrinsic defect was noted in the anterior stomach wall. A soft tissue mass continuous with the liver shadow was noted in three cases.

Pathology.—Primary carcinoma of the liver occurs in three forms: (1) Hepatoma, derived from the parenchymal liver cells. (2) Cholangioma, derived from the bile ducts. (3) Mixed type.

The hepatoma is the most common type (17 cases in this series). It is composed of masses of cuboidal cells with pink cytoplasm and large vesicular nuclei. The cells resemble those of normal liver parenchyma but are larger, grow in more irregular fashion, and

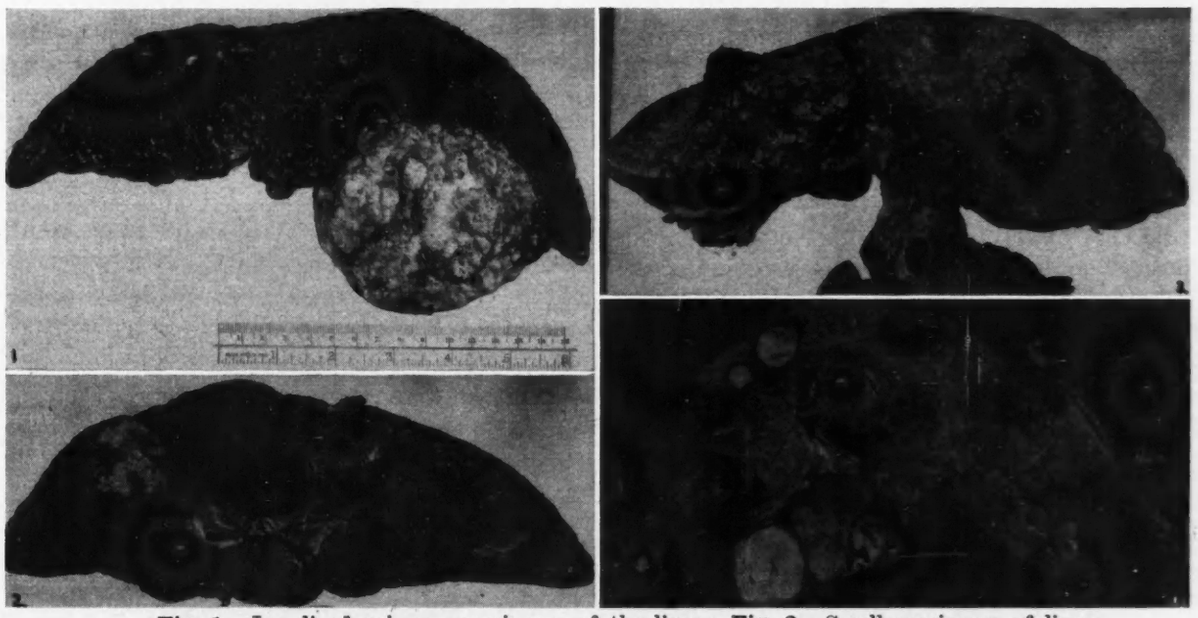

Fig. 1.—Localized primary carcinoma of the liver. **Fig. 2.**—Small carcinoma of liver in right lobe. **Fig. 3.**—Diffuse primary carcinoma of liver. **Fig. 4.**—Multiple primary hepatomata.

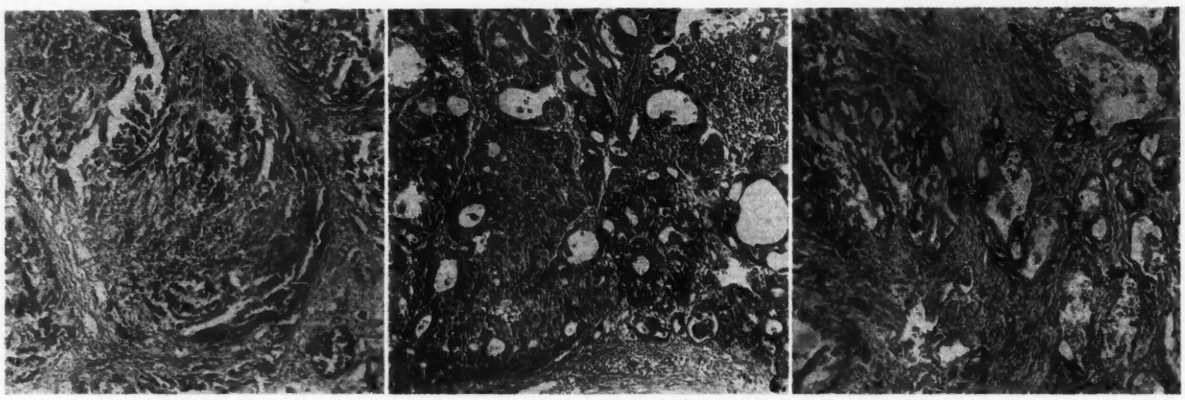

Fig. 5.—Primary carcinoma of liver—parenchymal cell type. **Fig. 6.**—Primary carcinoma of liver—biliary duct type. **Fig. 7.**—Primary carcinoma of liver—mixed type.

often contain mitotic figures. These growths tend to occur in areas of regenerative hyperplasia in cirrhosis. The tumours may be localized or diffuse or may rarely occur as multiple, solitary nodules. The cut section is sometimes bile stained and this characteristic may occur in metastases.

The cholangioma is said to occur more frequently in women. There were five cases in this series and four were in males. These tumours arise from the cells of the intrahepatic bile ducts and are adenocarcinomata with a tendency to form ducts and alveolar structures. They do not secrete bile. The cells are large with a pale basophilic cytoplasm and hyperchromatic nuclei.

The mixed tumours are composed of both parenchymal and biliary duct cells in varying proportions. They are relatively rare; two cases were found in this series. The site and extent of the neoplasm is vitally important in determining whether or not surgical treatment is feasible. In this group, 14 were in the right lobe, 6 in the left and 4 were diffusely scattered throughout the liver. It thus becomes apparent that only a small number would be amenable to adequate resection since the right lobe cannot be entirely removed. However, partial right lobectomy might be feasible in some instances. Metastases were present in 12 cases. They were found to be intrahepatic in 6 and extrahepatic in 6. The sites involved in order of frequency were regional lymph nodes, omentum, mesentery, lungs and adrenals. This does not represent a true incidence, because some of the cases confirmed by biopsy may have had metastases which were not visible at operation. Of the 9 cases which came to autopsy metastases were present in 5 and absent in 4.

Treatment.—To date the outlook has been hopeless in carcinoma of the liver. In our series, resection was attempted in two cases and both patients died from hæmorrhage and shock. These operations were performed in 1930 and 1935 and autopsy revealed complete removal of the growth in both cases. Recent advances in shock therapy and new methods of hæmostasis have made partial hepatectomy a feasible procedure and it is possible that some of these patients might be saved. When we consider that almost half the cases are amenable to resection the picture becomes more promising. The chief necessity is to establish the diagnosis early before spread occurs.

SUMMARY

1. Primary carcinoma of the liver is a relatively uncommon disease. Twenty-four cases have been observed at the Royal Victoria Hospital in the past 25 years.

2. The diagnosis is difficult but can be suspected in many cases on the basis of clinical and radiological findings.

3. From the pathological standopint, many of these tumours are localized, and in approximately half the cases, no metastases are present.

4. The prognosis has been considered hopeless but with new advances in shock therapy and hæmostasis partial liver resection is now feasible.

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SUTURE-LIGATION OF THE PATENT DUCTUS ARTERIOSUS IN INFANCY

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WE feel that the operative closure of the patent ductus arteriosus is best accomplished by the suture-ligation technique of Blalock. In the past three years, 75 cases of patent ductus arteriosus have been operated upon with one death and no recurrences. Encouraged by the simplicity of this operation, four cases of patent ductus arteriosus diagnosed in infancy were subjected to operation. Suture-ligation was performed at four months of age in two infants and at eight and fifteen months in two other infants. The diagnosis was made, in each case, by Dr. John Keith, cardiologist at the Hospital for Sick Children, Toronto. The lesion may be suspected in infants in which a murmur is present, cyanosis is absent and the heart is enlarging. The diagnosis of a patent ductus arteriosus in infancy is proved by the retrograde aortogram (see Fig. 1).

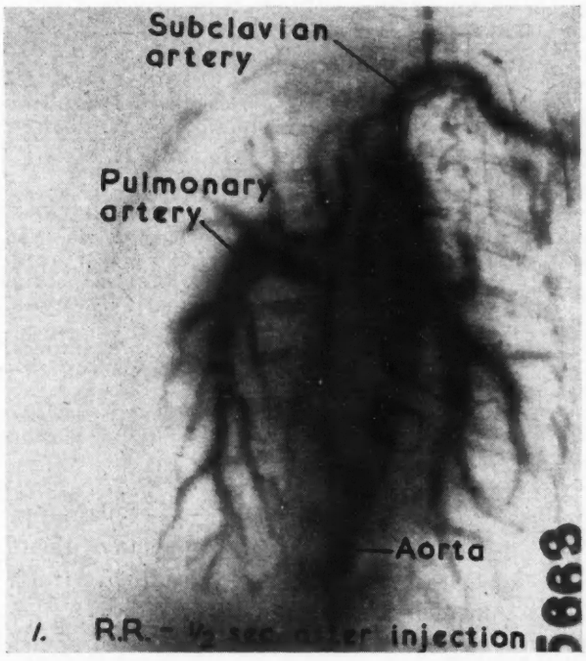

Fig. 1.—Aortogram demonstrating the dye entering the pulmonary circulation without passing through the heart.

The patent fetal ductus arteriosus probably closes functionally at birth or very soon after the lungs expand, and anatomical closure soon follows. Christie[1] examined 558 consecutive normal hearts and found that 95% of the infants showed the ductus arteriosus to be closed at twelve weeks. Closure of the ductus has been reported as late as two years following birth. Patency in the presence of an enlarging heart is definitely an indication for surgical intervention when one considers that in Hollman's[2] review of 28 autopsy cases from the literature, four were under the age of one year.

If the diagnosis is confirmed before the age of one year, the heart is normal in size and the general condition of the child remains good, it is rational to wait until the age of two or three before ligation is considered. If the heart is enlarging and the presence of a ductus proved by aortography, we feel thoracotomy is indicated. The operation in infancy is no more difficult than in later childhood and there are no technical differences. Following operative closure of the ductus the heart not only ceases to enlarge, but actually becomes normal in size (see Fig. 2).

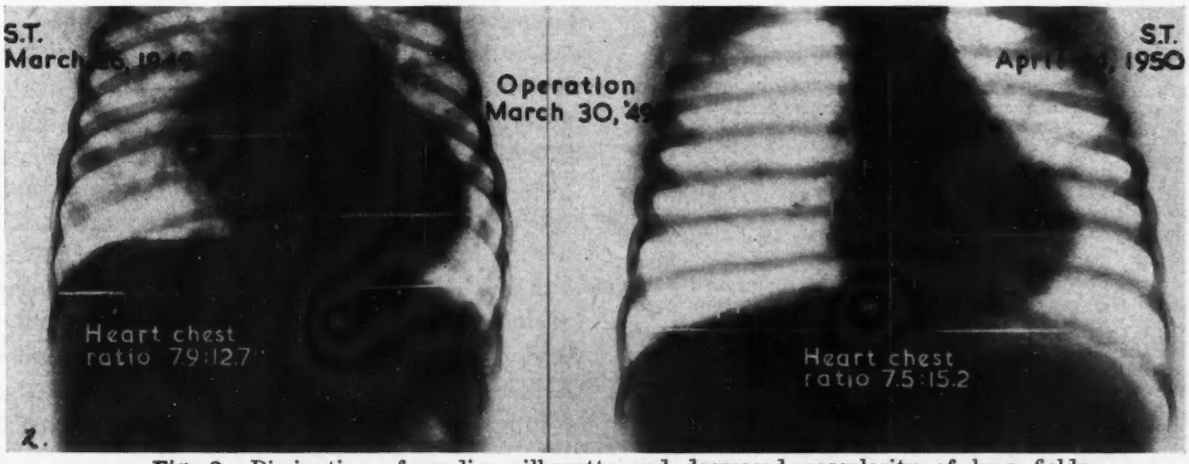

Fig. 2.—Diminution of cardiac silhouette and decreased vascularity of lung fields following suture ligation at eight months of age.

A summary of the cases operated upon appears below:

CASE 1

R.R., date of birth, February 1, 1949; suture-ligation performed May 6, 1949, at four months of age. On admission to hospital the infant had a history of frequent spells of gasping respiration followed by limpness and pallor. On examination, a systolic murmur was present over the precordium and an aortogram demonstrated the pulmonary vessels to fill from the aorta. The heart was enlarged. One year following ligation, at age of 16 months, no murmur was present and the child was gaining weight.

CASE 2

K.G., date of birth, December 9, 1948; suture-ligation performed March 18, 1949, at four months of age. The murmur was discovered at the age of three months, the child was not gaining or taking her feeding well. The heart was found to be enlarged and an aortogram was done which revealed the pulmonary vessels to fill directly from the aorta. Two years later, no murmur was present; the child was well and gaining weight.

CASE 3

S.T., date of birth, July 23, 1948; suture-ligation performed March 30, 1949, at eight months of age. A heart murmur was heard by an examining physician who advised admission to hospital, where the heart was found to be enlarged and an aortogram revealed filling of the pulmonary vessels directly from the aorta. One year following ligation, no murmur was present and the child was gaining weight. The cardio-thoracic ratio was decreased (see Fig. 2).

CASE 4

B.K., date of birth, November 17, 1948; suture-ligation performed February 10, 1950, at fifteen months of age. Murmur was present since birth and the heart was gradually enlarging. No aortogram was done, the diagnosis was made on the basis of physical examination. Seen two months later, no murmur was present and the child was gaining weight.

CONCLUSION

Suture-ligation of the patent ductus arteriosus is a safe procedure in infancy; if performed when the child is in cardiac failure it is life-saving. The diagnosis of this lesion before the age of one year is aided by aortography.

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To train enough professionals to keep all the people well by *curing disease* would involve an expenditure of so large a fraction of the income of any nation as to make the cure less attractive to the taxpayers than the disease. Indeed, if we tried to do the job today without our present knowledge of preventive medicine and public health, the situation would be hopeless. . . . The objective of the application of science to the problems of human biology is nothing less than the gradual reduction in the need for those experienced in the healing arts. Someone has said that the aim of preventive medicine is to prevent medicine! More soberly stated, one may say that as the profession of public health advances, the need for hospitals, physicians, and surgeons will diminish.—J. B. Conant.

INTRATHORACIC TUMOURS*

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IN the past the problem of intrathoracic tumours has been a particularly interesting one, but it has been made more interesting of recent years because of the mass x-ray surveys that have been undertaken throughout the whole country. Previously we depended largely on the patient's complaining of something before he consulted his physician. Now, very often the shadow of a tumour is found in a routine x-ray. The problem then is what should be done about these symptomless intrathoracic shadows. This paper is an effort to clarify the situation.

CLASSIFICATION

Pathologically, intrathoracic tumours may be extrapulmonary, intrapulmonary, or tumours of the thoracic wall.

1. *Extrapulmonary.*—(*a*) *Anterior mediastinum*: (1) Lymphomas. (2) Thymomas. (3) Dermoids and teratomas. (4) Thyroid adenomas. (5) Cysts (bronchogenic). (6) Lipomas. (7) Lymphangiomas and hæmangiomas.

(*b*) *Posterior mediastinum.*—(1) Fibroma, neurofibroma, perineural fibroblastoma, and ganglioneuroma. (2) Enterogenous cyssts. (3) Dural cysts.

2. *Intrapulmonary.*—(1) Fibroma. (2) Hamartoma. (3) Hæmangioma. (4) Adenoma. (5) Carcinoma. (6) Tuberculoma.

3. *Tumours of the thoracic wall.*—(1) Chondroma. (2) Fibroma. (3) Osteoma. (4) Giant cell tumour. (5) Angioma. (6) Myxoma.

Most of these tumours are self-explanatory but it might be well to enlarge on some of them. Lymphomas include the whole group of lymph gland enlargements such as Hodgkin's, lymphosarcoma, and lymphoblastoma. The thymomas may be either straight tumours of the thymus gland, or they may be lymphocytic tumours of the same gland. Many of the tumours in the anterior mediastinum are cysts, which may be either dermoid or teratomatous cysts, or bronchogenic cysts. The most common lesions in the posterior mediastinum are the solid neurofibromata, but here also may be found enterogenous cysts, and cysts arising from the dural canal, and extending laterally from there into the thoracic cage. Of the tumours arising in the lung itself of course the most common is the carcinoma. Adenoma of the bronchus is not a very common tumour and is seen much more frequently in women than in men. The fibroma of lung is similar to any other and is of itself

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of very little importance. With regard to hamartoma McDonald, Harrington, and Claggett[1] say: "We have confined the use of hamartoma of the lung to a specific type of tumour of the lung which has a characteristic morphological appearance. This is a solid tumour of the bronchus which consists of benign mesodermal, and benign epithelial elements". They also say: "although hamartoma of the lung is a benign slow growing tumour that usually is asymptomatic, we believe that it should be removed surgically". Tuberculoma of the lung is not a true tumour but is so similar to these other lesions radiologically that it is included in this classification to make it more complete. The tumours of the thoracic wall are largely self-explanatory.

SYMPTOMATOLOGY

Because of the frequency with which these patients are being picked up on routine chest x-ray, many of these patients have no symptoms whatever. Another small group have minimal symptoms which are found on questioning the patient, but which were not severe enough to cause them to consult their physician. Then there is a group of patients who complain of symptoms and these symptoms are mainly due to pressure or to irritation. Pain, shortness of breath, cough, difficulty in swallowing, or difficulty in breathing may be included in these symptoms. Generally speaking there is very little to be found on physical examination. The one exception to this might be in the neurofibromata where involvement of peripheral nerves may give a definite patch of anæsthesia or muscle weakness.

DIAGNOSIS

The exact diagnosis of this type of lesion is often difficult or even impossible without exploratory laparotomy. However, x-rays taken in several directions will show whether the tumour lies in the anterior half or the posterior half of the thoracic cage, whether it seems to be arising from the mediastinum, or in the lung. Pneumothorax may be an additional help to identify whether the lesion is intrapulmonary or extrapulmonary. Bronchograms may show whether the bronchial tree is blocked, compressed from the outside in the major bronchi, or compressed from the outside peripherally. Bronchoscopic examination is also indicated to rule out mucosal involvement of the major bronchi. It is possible that in some it is safe to watch the progress of the lesion radiologically over a period of time, but it is intended to show by the case presentations that this can be a very dangerous procedure.

TREATMENT

As suggested above there is frequently considerable difficulty in making an exact diagnosis, and this in itself is one indication for operative treatment. As will be demonstrated the possibility of carcinoma of the lung is ever present and there is also the possibility of malignant changes in other types of tumours. Although in many of these it is undoubtedly safe to wait, the indefiniteness of the diagnosis is always present, and it is generally considered wise to proceed with operative treatment. The approach depends on the location in the thoracic cage, but in the majority the usual postero-lateral incision is the most satisfactory one, and for this undoubtedly the prone position is the most satisfactory operative position. For extrapulmonary lesions excision is the treatment of choice. For intrapulmonary lesions either segmental resection, lobectomy or pneumonectomy may be indicated. Quite often it is possible by segmental resection and quick section of the biopsy to determine whether there is malignancy present; if not, the operation is completed. If the tumour is malignant then the total pneumonectomy can be proceeded with.

PRESENTATION OF CASES

It is proposed to demonstrate examples of many of the different types of intrapulmonary and extrapulmonary tumours along with their clinical history when available.

CASE 1

This was a man of 59 years who was seen in April, 1948, complaining of pain in the back and vague discomfort for about a month. No cause for this was found, and he had just recently had a coughing spell and coughed up a little blood. On investigation chest x-rays and bronchograms were done which revealed a tumour in the left lower lung field posteriorly. The bronchograms showed compression of the lower lobe, upper and forward. Bronchoscopic examination revealed blood coming from the lower lobe bronchus but there was no evidence of neoplasm. Exploratory thoracotomy revealed a large cystic mass which was compressing the lower lobe, but was entirely free from it. This was removed and that removal allowed the lower lobe to expand. Pathologically this cyst was a benign teratoma.

CASE 2

A woman of 50 years of age who had had a sub-total thyroidectomy many years before. She was perfectly well until about a year before examination when she began to have a dry non-productive brassy cough. During the year she also had intermittent hoarseness. She had no other symptoms and nothing could be found on examination. However, chest x-rays showed a mass in the upper mediastinum lying between the trachea and

œsophagus and pushing both to the right. Bronchoscopic and œsophagoscopic examinations showed no involvement of the mucosa in either organ and the recurrent laryngeal nerve was not paralyzed on either side. The diagnosis of sub-sternal thyroid adenoma was made and this was confirmed at operation. This was done by splitting the manubrium sterni and shelling the adenoma out from between the œsophagus and trachea. Her symptoms were relieved almost at once.

CASE 3

A woman of 42 years who had had no symptoms other than general fatigue and a slight shortness of breath. She had a routine x-ray which showed a large regular mass in the anterior mediastinum. This was removed through an anterior incision and pathologically was found to be a dermoid cyst. Her recovery was uneventful.

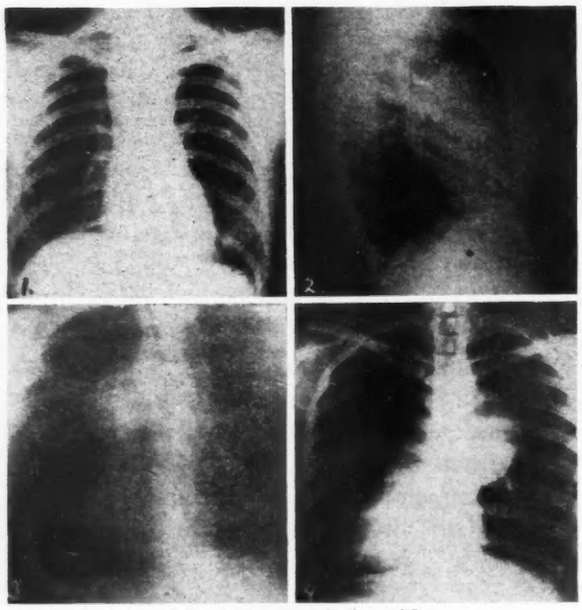

Fig. 1. (Case 2).—Substernal thyroid.
Fig. 2. (Case 3).—Dermoid cyst.
Fig. 3. (Case 4).—Oblique film—bronchogenic cyst.
Fig. 4. (Case 6).—Enterogenic.

CASE 4

A young man of 34 years who was x-rayed when he attempted to enlist in the army early in the war. A shadow was seen in the chest x-ray and he was not allowed to enter the armed services. He had no symptoms of any kind and repeated x-rays showed no apparent change in the shadow. However, it was felt that due to the size and the position of the shadow in the anterior mediastinum, it would be wise to remove the mass. Bronchoscopic examination was negative and the mass was removed in March, 1948, through an anterior incision. It was a thin-walled cyst, containing clear fluid and was classified as a bronchogenic cyst.

POSTERIOR MEDIASTINAL TUMOURS

Of tumours arising in the posterior mediastinum there are three examples.

CASE 5

This was a young girl of 16 years who had complained of a sharp stabbing pain in the right arm for four years, and dull aching pain constantly for one and a half years. This pain was down the inner side of the arm and forearm to the hand. She had also noticed this hand to be warmer than the left. Examination of the right arm showed an area of diminished sensation on the ulnar side of the arm, forearm and hand, and the grip was slightly weaker, but the reflexes were equal. X-rays showed a circumscribed mass with calcification at the level of the second rib posteriorly. A preoperative diagnosis of neurofibroma was made and this was confirmed at thoracotomy. The tumour was found to be associated with the first intercostal nerve and the sympathetic ganglion. Postoperatively the patient was relieved of her pain but had some residual ulnar anæsthesia.

CASE 6

This was a young girl of 21 years who had had hæmoptysis four or five times six months before examination. She was pregnant at the time but lost her baby at two and a half months, and in the succeeding investigation an x-ray of the chest showed a mass in the posterior mediastinum. At the time of the examination she was pregnant again and it was felt that something should be done about the mass in her chest before pregnancy had progressed too far. Consequently, following investigation which established no definite diagnosis, the thorax was opened and the mass was found to be a large cyst which was dumb-bell in shape and extended from the first rib above to the diaphragm below. It was excised with a moderate amount of difficulty. The pathological diagnosis of this cyst was a congenital cyst of the archenteron.

CASE 7

This was a young woman of 36 years who had complained of pain in the right chest for three years. She had been x-rayed at that time but the pictures had been thought to be negative. However, re-check plates a year before examination showed a shadow in the right chest which was found to be in the posterior mediastinum and seemed to have some eroding effect on one of the lower dorsal vertebræ. At operation this was found to be a cyst arising from the dura of the spinal canal which explained the evident erosion in the vertebral body. There was no nerve involvement. The cyst was removed and the base oversewn and covered with muscle and fascia, and the patient made a satisfactory recovery.

INTRAPULMONARY LESIONS — BENIGN

Of the intrapulmonary lesions there have been a fair number of benign lesions, although the great majority of course have been malignant.

CASE 8

The first of the benign lesions was found in a 61 year old man who had had substernal pain for two years, along with some shortness of breath. An x-ray taken when he first complained of the pain showed a circumscribed opacity in the right lower lobe, and later x-rays showed no change in the size of this mass. Other investigation was negative and at thoracotomy a hard fibrous mass was found in the right lower lobe. This was resected and the pathologist reported a hyalinized fibroma. The patient's recovery was satisfactory.

CASE 9

This was a 29 year old man who complained of periodic pain in the right side of his chest, since having multiple fractures of the ribs ten years before. For the previous four to five years he had had a chronic cough with a moderate amount of purulent sputum and more recently fatigue, loss of strength, and a slight loss of weight. Complete investigation of the gastro-intestinal tract was negative. X-rays of the chest showed a circumscribed mass in front of the right hilum. Bronchoscopy was negative. Thoracotomy revealed a firm mass in the right upper lobe and following lobectomy this was found to be a hamartoma. Microscopically this showed hyaline cartilage separated by trabeculæ of fibrous connective tissue with some adipose tissue scattered throughout. He made a good recovery.

CASE 10

This was a young veteran who was admitted to the Veterans' Hospital with recurrent hæmoptysis in 1945. X-ray showed a circumscribed area in the right lower lung field and further investigation was negative. Thoracotomy revealed a soft pulsating tumour in the lower lobe, and this was removed, revealing a hæmangioma of the right lower lobe.

CASE 11

This was another patient in the Veterans' Hospital, of 37 years, who was admitted in January, 1950, with pneumonia. He gave a history of four previous attacks of pneumonia and x-rays of his chest suggested an atelectasis of the superior segment of the right lower lobe. There was in addition a hilar shadow. Bronchoscopic examination was negative, and he was considered very seriously as a bronchogenic carcinoma. However, at operation the mass was found to be limited to the apical segment of the right lower lobe and a segmental resection of this segment was done. The mass on quick section was shown to be a bronchial adenoma and no further operative treatment was considered necessary.

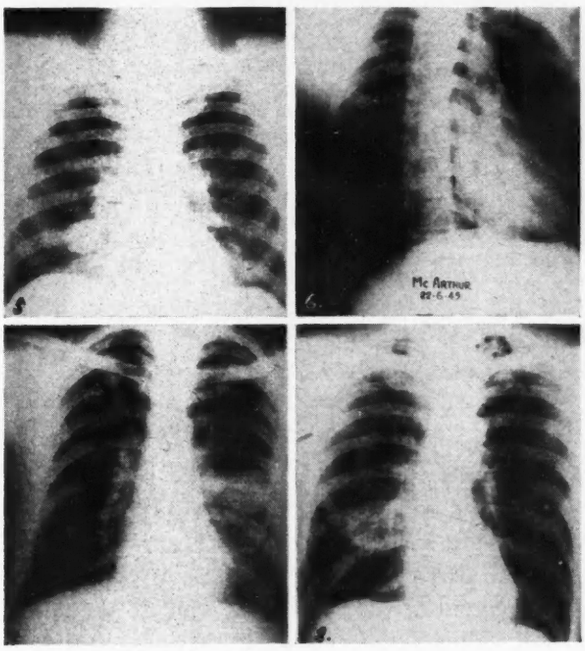

Fig. 5. (Case 8).—Fibroma.
Fig. 6. (Case 10).—Oblique film — hæmangioma.
Fig. 7. (Case 13).—Bronchogenic carcinoma.
Fig. 8. (Case 14).—Bronchogenic carcinoma.

INTRAPULMONARY — MALIGNANT

CASE 12

This was a woman of 47 years who was picked up in the chest clinic in 1945. She was complaining of a slight cough and sputum and an x-ray at that time showed a circumscribed hilar shadow. Diagnosis of bronchial adenoma was made and she was followed radiologically for two years. At the end of that time there seemed to be some slight increase in size in the mass and as bronchoscopic examination was negative, operative treatment was decided on. At the exploratory thoracotomy the mass was found to be in the right upper lobe, freely movable, with no peribronchial glands enlarged. The upper right lobe was removed and the mass was found to be a bronchogenic carcinoma. The lymph glands in the hilar region were negative on section. She developed a Friedländer's bacillus pneumonia postoperatively, so nothing further was done, and she lived for about two years and died of secondary carcinoma in the brain.

CASE 13

This was a man of 47 years of age who had complained of sharp pain in the left chest for three months. He had had a mild cough without any sputum for several years but this had not changed. There was some loss of energy and weight during the same three months. Physical examination was negative. Bronchoscopic examination was negative. The x-rays showed a circumscribed tumour in the left mid-lung field. A pneumonectomy was performed and pathologically it was a squamous celled carcinoma of the lung. He has now survived two years.

CASE 14

The patient was a man of 55 years who had a very slight cough and very little sputum for years, with a history of a bad chest cold three months before admission. Following this he had felt run down and a chest x-ray at that time showed a rather indefinite shadow in the right lower lobe. A chest x-ray taken a year previously had been called negative. Investigation was negative except for a very slight filling defect in the anterior basal branch of the right lower lobe bronchus, but in spite of the relatively few findings it was decided to operate on this man. The mass was found in the lower lobe which was slightly adherent to the middle, and both of these areas were removed. Biopsy showed carcinoma with negative hilar lymph glands.

TUBERCULOMA

There is another intrapulmonary lesion which is not actually a tumour but is classified as a tumour in the nomenclature, and that is a tuberculoma. This is a circumscribed chronic tuberculous reaction which is frequently unassociated with any acute tuberculous history or other physical finding.

CASE 15

A good example of that was seen in a war veteran of 57 years who was admitted to the Veterans' Hospital with pyelonephritis. In this routine chest film a round shadow was found in the right lower lung field. Other investigation was completely negative, then following his recovery from pyelonephritis he was advised to have this area of his lung removed. Accordingly, in December, 1949, a right lower lobectomy was performed and the pathologist reported a tuberculoma. He had an uninterrupted recovery and has had no further trouble since that time. This is fairly typical of this type of lesion.

DISCUSSION

It is quite apparent there is a marked similarity radiologically between many of these lesions. Certainly the difficulty of exact diagnosis is well demonstrated by this series of cases. Probably the separation into intrapulmonary and extrapulmonary can be done before operation, but generally speaking that is about as far as one can go. The exact diagnosis is made by the pathologist from tissue removed, either by quick section or by more careful study, and the exact operative procedure depends to a large extent on this report also. It would seem that the similarity radiologically between benign and malignant tumours in this series would indicate that all these localized circumscribed tumours

of lung or even of extrapulmonary tissue should be removed.

It is significant that more of these are being found on routine chest examination, and it is certainly true that the early diagnosis of carcinoma of the lung is made possible in this way if the adequate treatment is carried out.

SUMMARY

1. A relatively complete classification of intrathoracic tumours has been provided.

2. The symptomatology, diagnosis, and treatment of these various lesions has been discussed.

3. Representative cases of the more common types of intrathoracic tumours are presented and the importance of routine chest films has been emphasized.

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CASE REPORTS

KARTAGENER'S SYNDROME*

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Since 1904, there have been intermittent reports and reviews of cases presenting the combination of situs inversus, bronchiectasis and sinus disease, more recently known as Kartagener's triad or Kartagener's syndrome. The following is a report of such a case which came under observation in the medical service of the Victoria General Hospital, Halifax.

A white male, aged 49, was admitted to the Victoria General Hospital on December 2, 1949, with a history of productive cough for 40 years. He was one of five children, his father dying of a chest injury and his mother of unknown causes. Three brothers are alive and well, one died of diphtheria. There is no history of any member of the family having a congenital deformity.

The patient stated that he had had "a chronic bronchitis" with a productive cough for the past 40 years. He had pneumonia 30 years ago. An attack of measles in childhood was followed by a mastoid infection with subsequent mastoidectomy and resulting permanent facial paralysis. Nine months previous to admission, he suffered what he called a severe "cold", with marked increase in the amount of sputum. He had lost 12 lb. in the month prior to admission and had marked generalized weakness. At times he has had episodes of moderate hæmoptysis.

Examination revealed a man of moderate build, rather sallow complexion with his face drawn over to the left. The nasal septum was deviated to the left and the mucous membrane was congested. A few cervical glands were palpable. He was right-handed with some slight clubbing of the fingers. The right testicle hung lower than the left. Cardiac dullness and heart sounds were demonstrated to the right of the sternum and liver dullness was percussed in the left upper abdominal quadrant. These findings were confirmed by an electrocardiogram and a flat plate of the abdomen. Examination of the chest disclosed numerous, coarse râles over the left lung and some fine râles at the right base.

X-ray examination revealed the following findings:

1. Chest and bronchogram.—Dextrocardia, transposition of lungs and extensive bilateral bronchiectasis.
2. Sinuses.—All present; haziness of the left frontal, ethmoids and antra with thickening of the lateral walls of the antra.
3. Mastoids.—Sclerosis of right.
4. Gastro-intestinal tract.—Complete transposition.
5. Gall bladder and liver.—On left side.

Bronchoscopy disclosed purulent secretion coming from both main bronchi, more abundant on the right side; and also complete transposition of the lungs. Sputum examination gave a mixture of pneumobacillus of Friedländer and hæmolytic streptococci. No tubercle bacilli were found either on direct smear or by concentrate method.

During the first few days, this man produced from 6 to 7 ounces of purulent sputum daily. With aerosol penicillin therapy and postural drainage, the sputum was reduced to 1 to 2 ounces daily at the end of four weeks. During this period he also received 0.5 gram dihydrostreptomycin twice daily. The vital capacity was 2,100 c.c. or 52%. Surgical intervention was deemed inadvisable because of the extensiveness of the bronchiectasis. The patient was discharged from hospital feeling very much improved.

COMMENT

The first case of this nature to be reported was in 1904 by Siewert. It was in 1933 that Kartagener reported 4 cases and later 7 more of the syndrome which now bears his name. Olsen, in 1943, presented a total of 85 cases of true congenital dextrocardia, seen at the Mayo Clinic. Those cases in which the possibility of acquired dextrocardia was present were omitted from the survey. Of these 85 cases, 14, or 16.5%, showed definite clinical, bronchoscopic or radiological evidence of bronchiectasis. Of the 14 cases, 10 showed evidence of disease of the accessory nasal sinuses. In the same period under investigation, it was found that in less than half of 1% of all patients registered at the Mayo Clinic, a diagnosis of bronchiectasis was made. When this is compared with the above figure, the relationship between dextrocardia and bronchiectasis is more than a coincidence. Interestingly enough, it was found that 11 of these 85 cases had other congenital defects such as cleft palate, hydrocephalus, congenital heart disease, imperforate anus, etc., but none of the 14 cases with bronchiectasis had any other defects.

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In 1937, Adams and Churchill reviewed 23 cases of complete situs inversus admitted to the Massachusetts General Hospital over a 51-year period. They found that 5 of these cases had bronchiectasis as well as upper respiratory disease. The incidence of bronchiectasis in this series was 21.7% while the incidence of bronchiectasis in all hospital admissions over the same period of time was only 0.306%.

Richards, in 1944, reporting some cases, mentioned the congenital absence of the frontal sinus. Andrews, in a recent report, presented a case which showed congenital abnormality of the paranasal sinuses.

It is uncertain what rôle the transposition of the heart plays in the development of bronchiectasis but the high incidence of bronchiectasis occurring with dextrocardia would appear to be more than coincidental. It is claimed by several investigators that 80% of the cases of bronchiectasis occurring in childhood are congenital in origin.

Olsen makes the following comment: "The frequency with which bronchiectasis occurs among persons who have transposition of the viscera, its occurrence in twins and siblings and the increasing recognition of asymptomatic or dry bronchiectasis, would seem to indicate that maldevelopment of the bronchial wall may be a primary factor in the pathogenesis of bronchiectasis". In cases of bronchiectasis with no history of upper respiratory infection, or in cases with no history of bronchial obstruction, it is possible that congenital defects in the bronchial walls should be regarded as an etiological factor. Both congenital and acquired components probably enter into the production of the disease.

An interesting investigation into the genetics of transposition of the viscera by Cockayne in 1938 revealed that it behaved as a rare Mendelian recessive character. When monozygotic twins were born into affected families, it was found that transposition of viscera occurred either in both or in neither one. This did not occur in conjoined twins where the mechanism was different, one having transposition and the other being normal. This latter is probably nature's attempt to procure symmetry.

In the case being reported, it was felt that the upper respiratory infection was not primary but played a part in aggravating the underlying bronchial infection.

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I wish to thank Dr. C. W. Holland for his helpful criticism in the preparation of this article.

CARCINOMA OF BARTHOLIN'S GLAND

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This short paper is to record a case of carcinoma of Bartholin's gland.

Clinical history.—A nulliparous white woman of 41 years presented herself in March, 1950, complaining of backache, vaginal discharge, dyspareunia and a painful nodule on the vulva which bled on coitus. This nodule had been present for three months and was stated to be increasing in size.

On examination a small nodule was seen on the medial aspect of the right labium majus at the junction of the middle and posterior thirds. The mass was soft and friable and bled easily on palpation. The uterus was of normal size and retroverted. No masses were palpable in the fornices and the regional lymph-nodes were not enlarged.

The patient was admitted to hospital and, under general anæsthesia, the vulval nodule was excised by means of a wide elliptical incision and the operation area was closed by interrupted sutures. The uterus was curetted and placed in a position of anteversion.

The patient was discharged from hospital on the sixth postoperative day in good general condition with the operation site healing satisfactorily.

Pathology report.—Macroscopically the tissue measured 1.5 x 1.0 x 0.5 cm., was soft in consistency and of pinkish colour. In addition there was a small quantity of mushy, pink curettings.

Histologically the tissue showed a very disorderly adenocarcinoma-like structure infiltrating a fibrous tissue stroma. Gland formation was poor but the picture was chiefly remarkable for the large number of mucus-secreting cells. Many of these cells had the "signet ring" appearance characteristic of the Krukenberg tumour of ovary.

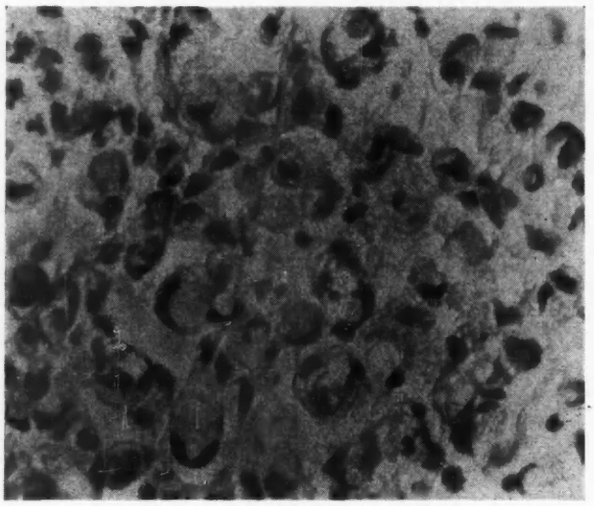

Fig. 1 (magnification x 645).

The uterine curettings showed normal progestin phase endometrium at about the 22nd or 23rd day of a normal 28 day cycle.

The final diagnosis was considered to be a mucus-secreting adenocarcinoma of Bartholin's gland.

The patient, as a resident of Saskatchewan, was referred to the Regina Cancer Clinic where a suitable radium implant was made on March 23. At the same time a further diagnostic curettage was done to exclude the possibility of the vulval lesion being metastatic. These curettings again showed normal endometrium.

Clinical review on June 1 and again on October 1, showed the treated vulval area to be well healed, with no evidence of local recurrence. The regional lymph nodes were not enlarged and pelvic and abdominal examinations revealed no abnormalities.

The general physical condition of the patient was good.

DISCUSSION

Carcinoma of Bartholin's gland can still be considered somewhat of a rarity. Simendinger[1] noted that there were only 38 recorded cases of primary carcinoma of this gland, of which 9 were of squamous type and 29 were adenocarcinomata. He added one more primary squamous carcinoma.

In 1941 McDonald *et al.*[2] reviewed the adenocarcinomata of the vulva encountered in the Mayo Clinic between 1906 and 1939. They found 32 tumours from 31 patients. Thirty of these were papillary adenocarcinomas, which they concluded had been derived from the apocrine sweat glands of the vulva. Willis[3] describes and illustrates a disorderly adenocarcinoma-like structure in a tumour from one of his cases and particularly mentions the mucus-secreting cells. Novak[4] states that primary carcinoma of Bartholin's gland is very rare and says that approximately 75 cases have been recorded in the literature. He agrees with McDonald *et al.* (1941) that the majority are papillary adenocarcinomata.

Primary carcinoma of Bartholin's gland must therefore be considered a tumour of rare occurrence and of these the majority are adenocarcinomas of papillary type.

The case here recorded of a mucus-secreting carcinoma of Bartholin's gland must be regarded as excessively rare.

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ABDOMINAL ANEURYSM*

J. Freundlich, M.D. and H. H. Pitts, M.D.

Vancouver, B.C.

The favourite site of luetic aortic aneurysm is the ascending part of the thoracic aorta. Its frequency decreases toward the ascending part and in the abdominal aorta arteriosclerosis is most frequently responsible for the formation of an aneurysm. However, cases have been published in which syphilis was the etiological factor.[2 to 6] It is the purpose of this communication to present the clinical and post-mortem findings of a patient who had an aneurysm of luetic origin of the entire aorta, thoracic as well as abdominal.

A 66-year old man was admitted to St. Paul's Hospital on February 12, 1947, because of severe shortness of breath especially at night. This condition began about two years prior to the admission and gradually progressed. He never suffered pain in the heart region; there was no history of rheumatic fever; venereal disease was denied. The patient was well built and moderately well nourished; his height was 5′ 11″, weight 153 lb. He had pronounced Cheyne-Stokes respiration, slight pallor and cyanosis of the lips. Left pupil was normal, reacted normally to light and accommodation; right pupil was deformed after an old iridectomy. The veins of the neck were slightly engorged, not pulsating; there was tracheal tug. In the precordial region a distinct bulge extended from the left sternal line to the anterior axillary line, upward to the second rib and downward to the sixth rib. There was a considerable pulsation of the entire chest. While the precordial region showed a depression during the systole, the right upper chest adjacent to the sternum revealed systolic heaving. The apex beat in the sixth intercostal space in the anterior axillary line was widened, increased, and resistant. On percussion the area of dullness extended also on both sides of the upper sternum about three fingerwidths. On auscultation a high pitched systolic and harsh diastolic murmur were heard over the entire heart with maximum intensity in the aortic region. The second aortic sound was loud and musical; no thrill was palp-

* From St. Paul's Hospital, Vancouver, B.C.

able. The heart action was regular, rate 112, gallop rhythm, blood pressure 180/90; Corrigan pulse, no pulse difference in the symmetrical arteries.

The upper and middle area of the left side of the abdomen also revealed a distinct pulsation. There was no manifest bulge but on palpation an expansile tumour the size of a grapefruit was found. No bruit was audible. Liver was slightly enlarged, no œdema. Deep reflexes were normal.

The radiological examination, revealed considerable enlargement of the cardiac silhouette, marked dilatation of the ascending aorta, and of the arch and fusiform enlargement of the descending part of the aorta (Fig. 1). Trachea showed some deviation to the right side. The electrocardiogram showed left axis deviation, T-wave was negative; Kahn was positive. Circulation time (arm-tongue) 28 seconds; venous pressure 18 cm. Urine, normal; x-ray (abdomen), well defined mass projecting into the abdomen from the dorsal area.

Diagnosis: aortic regurgitation, luetic aneurysm of the thoracic and abdominal aorta, cardiac decompensation.

The patient responded well to treatment and was able to leave the hospital in an improved condition. For the next two years he was under the care of his attending physician until January 23, 1949, when he was again admitted to the hospital with signs of severe congestive heart failure. The treatment had only temporary effect and he died on March 15.

Necropsy (H. H. Pitts).—On opening the pericardial sac, there is a moderate increase in pericardial fluid and a very markedly enlarged heart is revealed. It lies in an almost transverse diameter, the right auricular appendage and the tip of the left ventricle being on an almost horizontal line. The reason for this is a markedly enlarged ascending aorta which measures 10 cm. in diameter. The innominate artery also is markedly enlarged to 3 cm. in diameter. The arch is also markedly dilated in a more or less fusiform fashion and this dilatation is seen to extend throughout the thoracic aorta and the abdominal aorta to just below the cœliac axis and, at this point, there is a moderate constriction of the circumference and the common iliacs are relatively normal in diameter. There is a thinning-out of the under-surface of the right third rib in about the nipple line where the bulging aneurysm had impinged on the rib, but the periosteum itself is not actually eroded, and there is a concave depression in this area.

No other areas of osseous change are noted, that is, the thoracic vertebral bodies are quite intact and there is no evidence of rupture of the aneurysm in any area. The aorta was opened throughout its entire extent and is seen to be more or less uniformly dilated from the aortic valve down to a point just below the cœliac axis (Fig. 2). It measures in circumference as follows: ascending aorta 15 cm., arch 19 cm., descending 14.5 cm., and, at the point of constriction below the cœliac axis 6.5 cm. The abdominal portion measures 8 cm. in diameter. The walls are rather thickened and, in some places there are thinned-out areas where the wall would appear to be almost on the point of rupture. Only in an occasional area, and this in the more abdominal portion, is there some degree of thrombus formation on the wall, that is, there is none of the usual laminated thrombus formation seen in the lining aspects of the majority of aneurysms. There is a diffuse atherosclerotic process throughout the whole aorta on the intimal aspect and also ridged, roughened, so-called dog tongue-like striations which grossly suggest a luetic process. The orifices of the coronary arteries, however, are quite patent throughout and show none of the stenosis that is usually associated with luetic aoritis.

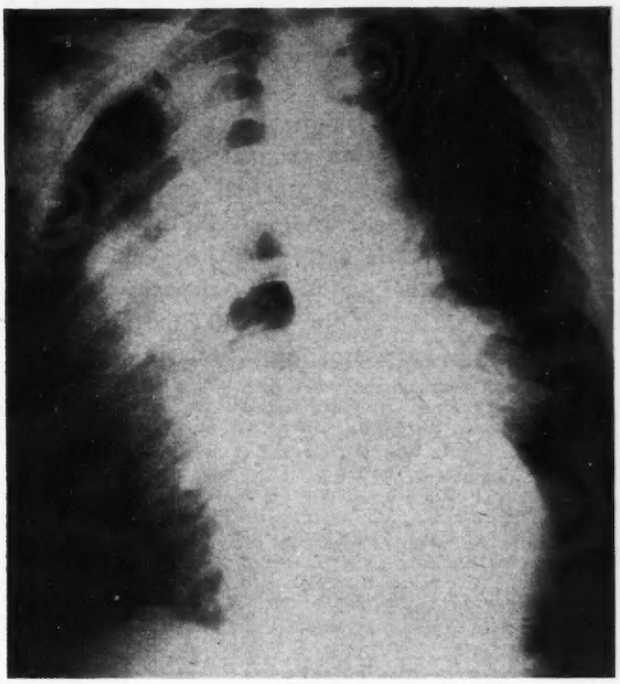

Fig. 1

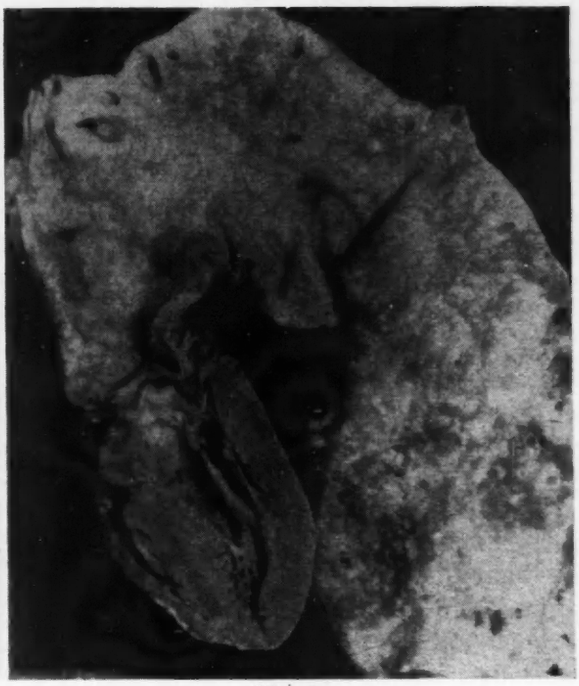

Fig. 2

Fig. 1.—Left anterior oblique position. Gross dilatation of the ascending aorta, the aortic arch and descending aorta, suggesting a fusiform enlargement involving all parts of the aorta in the chest. **Fig. 2.**—Mounted specimen showing marked generalized dilatation of entire aorta, the intimal "wrinkling" not being as well defined as in the fresh specimen. The hypertrophy of the left ventricular wall is well shown.

The heart, free of clot, weighs 900 gm., and the wall of the left ventricle is markedly hypertrophied to 3 cm. in thickness; the aortic valve measures 11 cm. in circumference and there is a relative insufficiency here and at the points of apposition of the valve edges, there is adherence and some degree of fibrosis. There is also a considerable degree of fenestration of the free margins of the valve cusps. The pulmonary valve measures 9 cm. in circumference and the mitral valve 11 cm. The right ventricular chamber is markedly compressed and actually, its capacity would appear to be less than that of the right auricle and, this apparently is due to the im-

pingement of the markedly hypertrophied interventricular septum on this chamber. The heart muscle is extremely firm and beefy. The coronaries are quite patent throughout and they show a moderate degree of atherosclerosis. The lungs are extremely œdematous and congested but, on section, show no evidence of any pneumonic process, only a hæmorrhagic œdema.

Microscopic examination.—Sections through the arch, ascending, descending and abdominal aorta were taken and in all a typical histological picture of luetic aortitis is seen. This is characterized by numerous perivascular lymphocytic infiltrations in the adventitia with smaller numbers in the media where fibrosis and areas of scarring are also noted. In addition well defined atherosclerotic changes with considerable calcific deposition are noted.

Diagnosis.—Massive luetic and atherosclerotic aneurysm of entire aorta.

COMMENT

The clinical picture had been dominated by the cardiac failure as manifested by gallop rhythm, Cheyne-Stokes respiration and œdema. But the striking signs were the pulsatory movements of the chest and abdomen, presenting a characteristic see-saw movement. While the precordial region showed a distinct inward movement during the systole, the right anterior thoracic wall, as well as the upper left abdominal region, showed a forceful systolic thrust.

The pulsations of the thoracic wall, known to elder clinicians, and more recently thoroughly investigated by Dressler[1] are often of great aid in diagnosis of some diseases of the heart, inasmuch as they are the result of the physiologic movements of the heart. Two factors are mainly responsible for the systolic depression of the chest wall. One factor is the diminution of cardiac volume during the systolic contraction, and another is the outflow of blood during the systole, from the thoracic cavity, causing a distinct fall of the intrathoracic pressure. Both factors, especially the fall of the intrathoracic pressure exert an aspiratory effect on the thoracic wall, the result of which is the systolic depression of the chest wall. This depression is especially pronounced when the volume of blood leaving the thorax cavity is increased as in the case of aortic regurgitation, when the unusually large stroke volume of the left ventricle is thrown into the aorta. While the aortic regurgitation in our case was mainly responsible for the pronounced systolic depression of the left thoracic wall, the forward thrust in the aorta and upper abdominal region was due to the forceful ventricular pulse transmitted to the dilated aorta.

The aneurysm itself did not cause much distress although its duration extended over a number of years. The common symptoms and signs of aneurysm were absent. Pain, usually a feature of an aneurysm (Osler[5]) was absent; there was no erosion of the ribs, sternum or vertebræ; nor pressure or obstruction of the neighbouring organs. The position of the abdominal aneurysm, as reported in the majority of cases, in the upper part of the abdominal aorta above the origin of the renal arteries, was also found in our case. The question whether the arteriosclerosis or syphilis is the main cause of abdominal aneurysm is often being raised by the authors. Scott[6] believes that the destruction of the media is more conducive to aneurysm formation than degeneration of intima or medial atrophy of arteriosclerosis. In our case the histological findings seem to support the luetic origin of this aneurysm although well defined atherosclerosis was also present and no doubt played its rôle in the extensive aneurysmal dilatation.

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PETECHIAL HÆMORRHAGES WITH THE USE OF GANTRISIN (NU-445)

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Gantrisin or NU-445 (3, 4-dimethyl-5-sulfanilamido-isoxazole) has the advantage of a relatively high solubility in neutral or even slightly acid urine. This obviates the need for concomitant alkali administration and it only rarely causes hæmaturia or crystalluria. It also has a low toxicity, and the only common side effects are erythema of the skin and gastro-intestinal upsets such as nausea, vomiting and anorrhexia. Hoffmann-La Roche Ltd. the manufacturers state in a private communication, that they know of only one previous case of petechial hæmorrhages with gantrisin and this was attributed to the concomitant use of penicillin. The following case illustrates this unusual complication.

Mrs. S.B., a 31 year old North American Indian was admitted to the Toronto Hospital for Tuberculosis on September 6, 1950, with a diagnosis of active Pott's disease of L.V. 1-4 with bilateral psoas abscess, and with active tuberculosis of the left midtarsal joint. The sedimentation rate on admission was 80 mm./hour (Westergren), Hb. 74%, white blood cells 6,900 with 72% neutrophils, 25% lymphocytes, 1% monocytes and 2% eosinophils. The red cells appeared smaller than average and many were poorly stained. A catheter specimen of urine was normal and grew neither tubercle bacilli nor secondary organisms on culture. The sputum was negative for tubercle bacilli on concentration and culture in 6 specimens. The Wassermann reaction was negative. Pus obtained by aspiration from both psoas abscesses grew tubercle bacilli on culture.

The patient was immobilized on fracture boards and the left ankle put in a plaster case. The psoas abscesses were aspirated from time to time as required. About the middle of September, 10 days after admission, she began to present symptoms of a mild cystitis with some scalding on micturition but no real frequency. A "sterile" specimen of urine voided under aseptic conditions on September 19 was reported as follows: pH 5.5, specific gravity 1.024, albumin slight trace, sugar negative, centrifuged deposit, gross pus with many epithelial cells and many bacteria; culture, hæmolytic *Staph. aureus*. Similar specimens also growing hæmolytic *Staph. aureus* were reported at the end of September and in October.

On November 6, the patient was started on a course of gantrisin in an attempt to clear up this chronic low grade cystitis. The initial dose was 2 gm. followed by 1.5 gm. six hourly, 4 doses per 24 hours. On November 15 the white cells had dropped to 4,600 but the patient showed no other signs of toxicity except for a transient flushing of the face after each dose of the tablets. On November 18, the 13th day of administration and when she had had a total of 75.5 gm., a petechial rash was noticed over the left subclavicular region, in the mucous membrane of the mouth, and over the arms. These hæmorrhages varied in size from pin point to 1 cm. in diameter. The white cells were found to be 3,900. The gantrisin was immediately withdrawn and penicillin 300,000 units of the aqueous suspension were ordered daily. On November 19, fresh petechiæ appeared, one on the soft palate being 2 cm. in diameter and this one burst and bled a little. On November 20, more petechiæ appeared over the lower end of the sternum and below and to the right of the umbilicus, but the patient had no other symptoms and felt perfectly well. The red blood cells were 3,500,00, Hb. 68%, platelets 260,000, white blood cells 5,000 with 73% neutrophils, 24% lymphocytes, 2% monocytes and 1% eosinophils. In view of this satisfactory response to treatment no additional measures were considered necessary. On November 21, fresh petechiæ appeared on the right calf but the white blood cells were 5,100. The stool showed a strong benzidine reaction in 1½ minutes. A "sterile" voided specimen of urine showed in the centrifuged deposit 3 to 4 white blood cells per high power field, many epithelial cells but no bacteria. By November 22, all petechiæ were starting to fade. The capillary fragility was tested with the petechiometer. At 20 cm. Hg. only one petechia was produced but at 30 cm. Hg. about 110 petechiæ were produced in a 1 cm. circle. This gave a normal capillary fragility of about 20 cm. Hg. The white blood cells on this date were 4,900. On November 23, the white blood cells were 5,800. On November 24, the penicillin was discontinued, the patient having received a total of 2,100,000 units. On November 25 the centrifuged deposit from a "sterile" voided urine showed no pus cells but had a few Gram-negative bacilli and culture of this grew *E. coli*. On December 1, the urine again showed no pus cells but many bacteria were present—possibly as a contaminant from the rectum. By December 4, 16 days from the first appearance of the petechiæ, they had all faded entirely.

COMMENT

Petechial hæmorrhages in the skin and mucous membranes are of course apt to occur with any of the sulfonamides and the outcome is not always as fortunate as in the present case. Here immediate withdrawal of the gantrisin resulted in the eventual clearing of the petechiæ and the return of the leucocytes to normal without further complication. The penicillin prevented any secondary infection taking place during the period of leukopenia. In the meantime the cystitis had been cleared up and the hæmolytic *Staph. aureus* infection eliminated. This reaction to the gantrisin was probably a simple idiosyncrasy to the drug. She does not have an allergic diathesis nor had she received any other drugs since her admission.

I wish to thank Dr. C. A. Wicks, Superintendent, Toronto Hospital for Tuberculosis, Weston, Ont., for his kind permission to publish this case, and Dr. H. E. Pugsley, Consultant in Internal Medicine for his advice.

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SURVIVAL OF UNUSUALLY PREMATURE BABY

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Quill Lake, Sask.

The following case is interesting as an instance of survival of an unusually premature infant.

Baby S.E. was born in hospital at Quill Lake, Sask., March 17, 1950, weight 2 lb. 7 oz.; length 14 inches. At birth the baby's colour was good and she had a healthy cry. Lanugo was still present on the face and the finger nails reached about halfway from the base to the finger tips. She had the appearance of a normal fetus of 6 to 6½ months.

The mother, aged 20, had a history of two previous pregnancies. Early in 1949 she had had an abortion at 6 weeks and an early miscarriage in August of the same year. She had been having prenatal care since February 13, but owing to an irregular menstrual history it was difficult to determine the exact date of confinement. On her prenatal visits there was nothing remarkable about her condition excepting occasional nocturia. She was taking Di Cal D with iron, capsule one t.i.d. She was admitted to hospital at 6 p.m. on March 17 with labour pains q.3 m. She was given the usual treatment for threatened miscarriage including progesterone and sedatives. The pains continued how-

ever and became more frequent and severe. She delivered at 9.00 p.m., L.O.A., a living female child as described above. There was no history of any untoward incident which might precipitate labour during her pregnancy so that one must look to her obstetrical history to explain this early onset of labour.

The baby was wrapped in cotton and placed in an Armstrong incubator, temperature 85 to 95° F. humidity 75. She was weighed and oiled with a minimum of handling for the first 48 hours. She was watched closely but no attempt was made to give her any nourishment. Excepting for a few minutes in the second day her colour remained good. There was some cyanosis then which responded very satisfactorily to oxygen. An adequate supply of mother's milk was placed at our disposal by the Mothers' Milk Bank at Saskatoon. After 48 hours she was given breast milk, 1 dram from a medicine dropper. The medicine dropper was used until April 12, after which a Breck feeder was used.

After the first feeding the amount was increased by 1 dram q.6 h. for the next 24 hours. Feedings were retained fairly well. She was then put on a two hourly schedule in amounts varying from 2 to 4 drams depending on how she retained the feeding. On April 3 the amount was increased to 5 drams q.2 h. At this time her weight was 2 lb. 1 oz. On April 12 there was another increase to 6 drams q.2 h. During this time she was retaining her feedings very well and there was an increase of 1 oz. in weight. Her condition was good and she was quite active. The amount of feedings was gradually stepped up until she was taking 1 oz. q.2½ h.

On April 27 weight was 2 lb. 5½ oz., length 15¾ in. Stools were yellow, pasty and of normal consistency. Abdec was added to her feeding beginning with 3 minims q.d. and increased to 6 minims q.d. The amount of bank milk was steadily increased until May 25, when she was taking 1½ oz. q.2½ h., corn syrup in small amounts having been added to the breast milk in the meantime. At this time she weighed 3 lb. and we put her on a feeding of breast milk 75% and formula (Carnation 5 oz.; water 15 oz.; corn syrup 1 tbsp.) 25% on a 2½ hourly schedule. At the same time we began to give her orange juice ½ dram with an equal amount of water daily. This was steadily increased until she was getting 2½ drams in water when she was discharged. Pure cod liver oil was also added to the feedings beginning with 5 minims t.i.d. and steadily increasing to ½ dram t.i.d. at discharge. For the remainder of her stay in hospital we also gave her elixir fergon beginning with 2 minims in her feedings four times daily. This also was increased until she was getting 15 minims four times daily at discharge.

The feedings were gradually changed from breast milk and formula to artificial feeding, and by July 5 the baby weighed 5 lb. 6½ oz. When last seen on September 27 she weighed 10½ lb. and appeared to be developing normally, both physically and mentally.

I wish to thank Miss Katherine Dyck, R.N., Matron of the Quill Lake Union Hospital for her untiring efforts on behalf of this baby.

The Section of Anæsthesia, Ontario Medical Association, wishes to announce a two-day clinical meeting at Peterborough on March 16 and 17, 1951.

Dr. Virginia Apgar, Anæsthetist at Presbyterian Hospital, College of Physicians and Surgeons, Columbia University, New York City, will be the principal Speaker.

Details of the program and arrangements for accommodation will be mailed to members of the Section.—J. A. Vining, Secretary, Section of Anæsthesia, Ontario Medical Association.

SPECIAL ARTICLE

THE ROLE OF THE HUMANITIES IN MEDICAL EDUCATION*

H. B. Van Wyck, B.A., M.B.(Tor.), F.R.C.O.G., F.R.C.S.[C.]

Professor Emeritus in Obstetrics and Gynæcology, University of Toronto, Toronto, Ont.

[*Je désirerais sincèrement vous présenter cette conférence en français mais je suis bien convaincu que personne ne me comprendrait, moi-même le premier. Vous me permettrez de continuer en anglais.*]

May I begin by acknowledging the honour you have conferred upon the obstetricians and gynæcologists of our College by according to one of us the privilege of addressing the annual dinner. An obstetrician should be able to deliver any reasonable presentation, but, alas! there is no guarantee against stillbirth. All that any practitioner may promise, all that justice demands, is the exercise of reasonable care and skill.

The gracious introduction that I have received from the President was so generous that I might suspect a case of mistaken identity. I am reminded of the story of a drunken alumnus at the football stadium. High up in the stand, he kept calling, "Hey, Gus"; and, each time, a man down in the third row would ceremoniously stand up and doff his cap. After many calls of "Hey, Gus", the gentleman in the third row shouted in a thick voice, "Now quit yelling at me! I am tired of standing up; and, besides, my name ain't Gus!" However, although my name isn't Gus, I find myself on my feet before you and in the stout words of Martin Luther, "Hier stehe ich. Ich kann nicht anders thun".

The selection of my subject took some thought. I suggested to the Council "Occipito-Posterior Position". The Council felt that such a presentation might take too long. I promised to shorten the whole thing by advocating earlier rotation and to entitle it "One Good Turn Deserves Another". The Council which had not yielded at that point to certain importunities of the gynæcologists did not wish to commit themselves for such a small reward. They were not amused. I had to agree—one has to with that obdurate Celtic master of irony, the chairman. You remember the Scotch Minister's prayer; "God grant that I may be always recht for I'm an awful hard man to turn". My proposal did produce a slight smile on the face of the Vice-president in Medicine. He is not without a rudimentary sense of humour, because all Scots have a sense of humour: of course, because—it is a gift. Yet I believe I know why all Scots dislike jokes about themselves: it is because they are naturally always at their expense.

* Address delivered at the Annual Dinner of the Royal College of Physicians and Surgeons of Canada, Montreal, December 12, 1950.

So I find myself with a subject of which the importance is balanced only by its difficulties. Varsity sometimes show up best when the going is difficult—as witness a certain recent muddy afternoon at McGill. I hesitate to introduce a serious topic when I realize that you have already had a pretty strenuous time listening to one another through two days of scientific sessions. If there are any of you who feel you cannot take any more, I would advise you to leave at once. I only wish I could sneak out with you. I promise to keep in mind two potential dangers of the after dinner speech—length and sobriety. You may have heard of the after dinner speaker who was droning on at too great length when the toastmaster was embarrassed to see an auditor within reach of the head table asleep and beginning to snore. He quietly reached out his gavel and tapped the sleeper lightly on his bald head. The offender woke up with a start, realized where he was, and then said, "Hit me harder, I can still hear him". Which recalls that when a popular preacher was asked if it bothered him to see persons in his audience take out their watches, replied: "Not unless they begin to shake them". Some after dinner speakers indeed would be better served by a calendar than watch.

To return to the metaphor supplied by midwifery. My subject which I am endeavouring to bring into the light—The Rôle of the Humanities in Medical Education—is at present alive, although it is threatened by the complication of technology and the too exclusive preoccupation of our schools with scientific method; threatened by a materialistic outlook, which tends to forget that man is an organism endowed with free-will, vibrant to, and thirsty for the spiritual values of life; and threatened by purblindness to the limitations of the natural sciences. In spite of these dangers which could extinguish the life freely given by the humanities, one must have faith that these essential elements in education may continue to survive with unextinguishable vitality.

One must pay tribute, especially here in Montreal, to the classic address on the Relation of Humanism to Science. "The Old Humanities and The New Science" was delivered on May 16, 1919, to the British Classical Association by the greatest humanist in the medical profession of our day, Sir William Osler. May I quote the last moving few lines:

> "Is not the need of individual reconstruction the Greek message to modern democracy? . . . With the hot blasts of hate still on our cheeks, it may seem a mockery to speak of this as the saving asset in our future; but is it not the very marrow of the teaching in which we have been brought up? At last the gospel of the right to live, and the right to live healthy, happy lives, has sunk deep into the hearts of the people; and before the war, so great was the work of science in preventing untimely death that the day of Isaiah seemed at hand, when a man's life should be 'more precious than fine gold, even a man than the golden wedge of Ophir'. There is a sentence in the writings of the Father of Medicine upon which all commentators have lingered. . . . the love of humanity associated with the love of his craft!—philanthropia and philotechnia—the joy of working joined in each one to a true love of his brother. Memorable sentence indeed! in which for the first time was coined the magic word 'philanthropy', and conveying the subtle suggestion that perhaps in this combination the longings of humanity may find their solution, and Wisdom—*Philosophia*—at last be justified of her children."[1]

Thirty years have passed since this immortal address was given and again the hot blast of hate is upon our cheeks. The combination that Osler desired was more science for the humanist and more humanism for the scientist. What would his bitter comment be on the tendency today to more science and less humanism as he would see it on this continent? Let me quote a typical statement from a prominent American educationalist which appeared in 1940:

> "The student and the citizen need to absorb the scientific attitude, to master the scientific method of thought, and to understand the basic concepts of the sciences. Only thus, delving beneath the superficial and avoiding the burden of the technical, can they be ready to read further and to understand in the decades to come what science is doing and can do. Only thus can their own intelligence be called into play."[2]

This dirge has been chanted by a large chorus of scientific departments in our universities. Sir William would be the first to exclaim against the implication that there were no intelligent men before Science was invented. He might have paraphrased Butler's poetical exclamation which had its unjustified locale in this city, "O God! O America!"

Intellectual climates sometimes change more rapidly than political forces. As recently as Osler's day, science conceived matter as genuine substance. Today the solid comfortable world of the materialist has withdrawn behind a delicate screen traceried by mathematical formulæ. These can tell us all too little of reality. Osler would view with alarm the present tendency of Science to be either a philosophy or a religion; he would be the first to note that as a moral dynamic without which our Western World may not survive, science has failed.

There are two main problems confronting the medical educationalist. First, the selection and balance of the scientific subjects in an undergraduate curriculum already œdematous with an overload of detail. The second problem—the one with which I have the temerity to deal this evening—is the addition to the already groaning camel of the elements to make education liberal without the imposition of the fatal last straw of time and expense.

How this latter is being done or how it should be done, I am not here to discuss. I shall con-

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1. Sir William Osler. The Old Humanities and the New Science. Houghton Mifflin Company, 1920.
2. This quotation occurs in "Science Is A Sacred Cow", by Anthony Standen. E. P. Dutton & Co., p. 22, 1950.

fine myself to why it must be done. This College, representing the Art and Profession of medicine at its highest standard in Canada, is surely a splendid forum for such a thesis.

I have already mentioned three obstacles in the way—the crowding and urgent demands of the technological advance, the materialism that tends to misdirect modern life, and the all too prevalent indifference to the real purpose of education. This latter is the heart of our subject. Much has been written lately in an effort to define education. It has been defined as knowledge of the things that matter most. Surely then the glorified splendour of a Gothic cathedral built in adoration of the Most High matters as much to the spirit of man as the blood level of creatinine matters to his body? Another definition of the means and ends of education is Whitehead's inspiring phrase, "the habitual vision of greatness"—a slogan which Sir Richard Livingstone considers the most profound saying about education since Plato: "the habitual vision of greatness" without which moral education is impossible—a clarion call to all teachers. Is the sublimity of a Beethoven or a Shakespeare dwarfed by the labours admittedly Herculean of an Einstein or a Pasteur? These questions, not rhetorical, demand an answer. What reply we give depends on our sense of life's values, upon what does matter most.

That education has two objects is admitted. Training for a living, or put more nobly and truly, training for service and citizenship is one aim; the other is training for a life. Man can not live by bread alone. Our profession has always kept this ideal before it—that the care of the body is no more important than the care of the soul. How futile to preserve life if it can not be made abundant! And what end is served by making a living if one does not know what to do with the living when one has gained it? Our boasted increase in longevity has accentuated the need of an answer. Very many of the problems of geriatrics would be solved by the solace of the humanities.

And now follows the ineluctable query. Can the natural sciences if left to themselves produce the abundant life? To us who ask this question and the optimistic Bacon who thought the increase in material science would inevitably bring complete happiness to man, the subsequent experience of three centuries of unprecedented material advance has provided stark disillusionment. To humanity the material sciences when untempered by humanism have promised bread but have brought only a stone.

What are the limitations of natural science? Some naïve souls, including medical students, believe that when they embark on scientific studies they leave all doubt behind. May I ask them what science they mean? Today's or tomorrow's? Natural science disavows its creeds as blithely as a broker ignores his mistaken advice or as a crab sheds its exo-skeleton. *Sic transit gloria mundi*—which the school boy whose Latin was optional translated, "Here Monday and away Tuesday". Now a medico should be the first proudly to point to the benefits which the natural sciences have conferred upon mankind. He has been able to bring the aid of the biological sciences to the bed-side. The brave result is the last one hundred years of scientific medicine. These sciences as handmaidens to medicine have been the only ones as yet not dedicated to destruction.

But just because we can smugly point to the greatest achievements of science, we should keep before our students what true science is and is not. If we do not, the products of our technical schools tend to lose faith in humanistic and religious values. In their place—as the mind of youth abhors a vacuum—they substitute a new naïve faith less coherent and beautiful than the old and based on the shifting sands of scientific method. These students of ours become the victims of a new superstitious scientism, itself the creed of amoralism. If this is the end of the academic road, instead of a wise graduate, we have a sophomore mentality never to come of age. He then truly exemplifies the child's definition of an adult—one who has stopped growing except in the middle. For the truth is that science can never invade the realm of the ultimate values or discover the soul. To acknowledge these limitations is to escape spiritual blindness. Science deals with those things which can be measured. It can not deal with the qualitative aspect of things. It gives only mathematical abstractions. An abstraction can never contain the whole truth. Such abstractions are the least important aspects of reality except for practical purposes.

One may give in illustration of the limitations of the scientific view, a celebrated example cited by Sir Arthur Eddington. He considers the case of an elephant sliding down a grassy hill side. What account can the physicist give of this? He wishes to know how long it will take the elephant to reach the bottom. For the elephant, he takes two tons, for the sloping hillside, an angle of 60 degrees, for the resistance of the turf, a coefficient of friction. Now instead of the delectable elephantine journey, he has an equation without remainder. 2 tons x 60/90 x coefficient friction (with certain modifications dear to the heart of the mathematician) = to pointer readings on the dial of his watch. This equation is the result of a mathematical abstraction of the elephant and the hill. It is far from a complete picture of the delightful drama, most of which is lost: the excitement and joy of the elephant; the feel of the cool turf on the not insensitive rear of the pachyderm; the dismay when an unexpected bramble bush is encountered on the way down; and the apprehension of parental

rebuke from his elders if the end of the trip lands him at the edge of a precipice. These elders, if uninstructed in modern psychology, may be tempted to point out the folly of embarking on a perilous adventure, the end of which is unforeseen. None of these exciting and romantic phantasies appear in the scientific formula.

Science deals with a closed world of measurable abstractions. These alone are amenable to treatment by its methods. Even its practical advantages are over-rated. For instance, a pressure cooker can cook spinach in two minutes which only means that one has to eat the beastly stuff fourteen minutes sooner. And a great many practical things in medicine can be done without recourse to scientific explanation. For students learning how to hold a baby, the best instruction is to point out that the nascent mammal requires the same grip as a cocktail shaker. Miss Jones had a better grasp of this complex world in which we live. The physics professor, with his single track mind, intent on his mathematical abstractions, asked Miss Jones what happened when a body was immersed in water. Miss Jones with a more catholic understanding replied, "the telephone rings".

The scientist who is not at the same time a humanist, may believe that the law of thermodynamics and the autonomic nervous system can explain all the thrill in poetry as well as the blush on his sweetheart's cheek; he believes that scientific sociology can reconcile the differences between nations; if he is a psychologist, he enthusiastically discusses the soul, the existence of which he denies; and, if a behaviorist, he tends to psychoanalyze the speech of his best friend until its meaning disappears entirely. One may well suspect that such a dehumanized human eats concentrated vitamins and regulates his domestic felicity by synthetic hormones. This scientific ideal, the portentous nonsense of a scientistic robotism must not be held up to our medical students as the goal of education. A mechanistic world without the humanism of the arts and religion becomes a world of horror divided between a scientific priesthood robed in infallibility and laymen who are their dupes. John Dewey, who is a devout communicant in the cathedral of scientism has stated, "the future of our civilization depends on the widening spread and the deepening hold of the scientific habit of mind".[3] There would, however, seem to be wisdom in the following sentence penned by Oliver Wendell Holmes: "Science is a good piece of furniture for a man to have in an upper chamber provided he has common sense on the ground floor".[3] To be modern, one might change the phrase "upper chamber" to the "service room".

Medical students must be provided with breadth of vision and wider horizons. One might recall the college rugby star who thought the Greeks must have played baseball because he had heard much about a famous Homer; and there was a neuro-psychiatrist who thought that Homer had written the "idiot and the oddity"—an ineptitude similar to that of the student of history who was asked in a lecture on Greek culture to name two ancient sports and replied, "Antony and Cleopatra".

A survey of the whole panorama of existence discovers the fields occupied by science to be much narrower than they appear to those who have not ventured beyond their limits. The values of life can not be mastered in terms of more or less; they are not amenable to scientific method. The scientist can give no account of music, poetry, the arts, or the spiritual aspirations of man. Science classifies on the basis of common qualities. Science defines the whole as the sum of the parts. Therefore it attempts the impossible in a complete description of living organisms, and, especially, of the personality of man.

What! you may ask, is not psychology the new science to explain the soul of man? Some have questioned the right of psychology to call itself a science by asking where are its data verifiable by experiment.[4] Certainly to the physician, the observed data of psychic analysis are utterly variable and unpredictable, to which the canons of science do not apply. The factors essential to us are the mental processes behind the observed data. Psychology interests itself in the observed phenomena of human behaviour from which it often unwarrantably assumes causation. One might point out that the more important aspect of human behaviour is not its causes but its values. Inasmuch as modern psychology claims to be scientific, it must be silent on values.

Some modern psychologists have discovered with an air of perplexed wonder what not even Hippocrates or Aristotle ever doubted—that soul and body have the most fundamental influences upon each other. Having observed this close relationship of mind and body, some of these pseudo-scientists with confused minds have assumed that they are the same thing. Out of the relativity of values thus engendered and now fashionable emerges a new morality which seems very much like the old immorality.

Preferably for the mature postgraduate student and not the immature entrant to University, there is doubtless good in objective psychology, especially in so far as it remains physiological, but this new scientific approach to the old problems is no substitute for the humanities. It is my belief that one gains more insight into the personality of man from great literature than from all the discordant schools of psychology.

3. This quotation occurs in "Science Is A Sacred Cow" by Anthony Standen, E. P. Dutton & Co., p. 35, 1950.

4. Walshe, F. M. R.: The Linacre Lecture, E. & S. Livingstone Ltd., Edinburgh, 1950.

If many of the hours of psychology we inflict upon medical students were given to history, literature, and poetry, these students would be better prepared for psychiatry and the psychosomatic approach to medicine. In other words, they would gain a clearer understanding of the human souls who will be their patients. This modern rage for psychology, with the comparative neglect of man's imaginative creations, will be regarded by posterity as we today regard the mediæval indifference to experimental medicine.[5]

Science alone can not save. Whence the broader field of education? Where may we pursue the values of life to which all else is means only? The ancients taught 2,000 years ago that wisdom was the supreme good, that wisdom was to be gained by the contemplation of truth, beauty and goodness; and that these were to be found in studies that concern the soul of man: philosophy, history, art, religion. For the problem of intellectual blindness born of scientism is closely akin to the problem of religious indifference, both sired by an old devil called materialism, and long known to the human race as the enemy of both the mind and the spirit. Hence the humanities in education should keep alive the unquenchable sense of religious values in man. Their function is to teach us the things which matter most. What are those things which matter most? And what can science tell us of these? What is the relation of mind and body? What was the beginning and what will be the end? What is human destiny? What of human will, love, hate, justice, liberty, ethics, conduct? The spellbinder of the laboratory is silent. These are undeniably difficult problems. We might be content with less—with a physico-chemical analysis of Hamlet or a formula for its creation; with a prescription for the murals in the Sistine Chapel, the carvings of the Parthenon, or the fair loveliness of the Unfinished Symphony. If for these we know no appeal but to science, we remain with empty hands.[6]

That the realms of pure science which knows not the values would prove to be insufficient for the nurture of personality, if spiritual values were to survive, has been the burden of much dire prophecy. It is wrong to base an estimate of man's future welfare on the degree to which his material surroundings are capable of improvement. This fallacy had its clearest and most enthusiastic announcement in the New Atlantis of Bacon at a time when the scientific method had its rebirth with the Revival of Learning. Bacon assumed that man would be both happy and wise as he perfected his mechanical environment; that his welfare depended on the ingenuity of his machinery; that his control of the material world would bring contentment and satisfy his deepest aspirations. This has proved a delusion, obvious now even to the most naïve materialist. Science has brought the promised increase in power. Man, failing to keep pace with the evolution of spiritual and moral development, finds that his triumph is empty. His dreams of Utopia have changed to visions of horror and despair. Modern man has been brought to the brink of annihilation by a science unchecked by the reins of humanistic thought. He must now perforce heed the voices from the wilderness that have never ceased to echo down the centuries. The way of repentance is the abandonment of materialism and a renewal of the zest to seek among the temporal deceits of external nature the road to that invisible realm of reality—a moral universe.

This requires a harmony of the intellect and the spirit, a harmony never yet fully attained by any of the great historic civilizations but an achievement upon which the future of humanity depends. Our Western Christendom did make a significant beginning in the attainment of spiritual and intellectual independence in the age of St. Francis and St. Thomas Aquinas; but since the Renaissance, the religious tradition which is the essential matrix of our civilization has gradually receded from our vision, and in the task of material organization the Western world has lost touch with it, with a resulting disunity of the moral and spiritual order. Nevertheless, the culture of the West derives its distinctive character to the degree to which intellectual and spiritual harmony has at times existed; and only through the co-operative harmony of both forces may our West survive in the realization of our latent but untried potentialities and build a new type of civilization equally removed from the fruitless and inactive apathy of the Ancient East and the feverish but aimless materialism of the modern West.[7]

No generation of man since the rise of civilization has lacked prophets. Large is the honour roll of those who have recalled man from the idolatry of matter and have pointed to the design, meaning, and purpose, of the universe. It contains, among the immortals, countless humbler folk whose thoughts and lives were based on the higher concepts of the spirit. Of these, the voices are stilled. Heard once again with greater clarity because of the shadow of futility and terror across the path ahead, are the words of Plato, inspired to find

5. Some of the ideas in this paragraph were stimulated by the witty and brilliant chapter on psychology in Anthony Standen's "Science Is A Sacred Cow", E. P. Dutton & Co., 1950.

6. Dixon, W. M.: The Human Situation, p. 56. Longmans, Green & Co., New York. This paragraph is paraphrased from this reference where the argument is developed in a passage of great beauty and power.

7. This paragraph was suggested by the last chapter of Christopher Dawson's "Religion and Progress", Sheed & Ward, London, 1949, and is its central theme.

beyond the visible but transitory world of sensation, which his instinct distrusted, the unseen ideal; of Socrates, who looked within the soul for the values of life, and two thousand years ago, rebuked the modern psychologist so pre-occupied with seeking out the causes of human behaviour by external observation, that he seems blind to its purposes and values; of the Nazarene who asked, "what profiteth it a man if he gain the whole world and lose his own soul?"; of Erasmus whose tortured cry rings out across time as if uttered yesterday: "See what the world is coming to—rapine, murder, plague, famine, rebellion, no one trying to mend his own life". Is not this what Osler meant by individual reconstruction? To none of these seers was the peril of a technocratic society such as ours a matter of experience; but to such is truth revealed in prophetic vision, whose radiance reaches the infinite recesses of human personality.

The redemption of man's folly may well come from many sources, among which education, while far from a complete means of salvation, provides at least a practicable and essential stream of influence. Uncritical devotion to science fosters a deterministic and materialistic view of our universe, without the meaning that only the concepts of the values can give. Such a concept of a world without moral purpose finds a congenial abode in totalitarian and despotic states. For this unbalanced view of life the antidote may be found in the humanities; philosophy which seeks to interpret the concrete by the abstract, the apparent by the real, the transitory by the eternal; history which records the reaction of the race to the experience of life; literature which preserves man's highest aspirations and reveals to his fascinated gaze his own potentialities for good and evil; music and the arts which justify his creation and give promise of high destiny; and religion which binds these nobler concepts into the acceptance of a universe of values—a moral universe—and defines a way of life that has a meaning.

There may be some who regard the inclusion of religion in the list as revealing a confusion between "humanitas" and "divinitas", between naturalism and the transcendental. In opposition to the narrower scientific view of the universe, the distinction might appear to be of little importance, as both humanism and the transcendental elements of religion surely stand four square against materialism. It should be recalled that from the time of the Renaissance, in a Europe divided by sectarian controversy, humanism has provided the only common ground upon which the different churches and schools of philosophic thought have been able to meet on equal terms and with common ideals. It may also be noted, in Christopher Dawson's words, that although humanism has been instrumental in the secularization of our Western culture;

> "It was by no means altogether irreligious. In this respect I think the Humanists have been ill served by their modern apologists and admirers, with the result that the current popular conception of the Renaissance and the humanist culture is erroneous and one-sided. It is not in Poggio or Machiavelli or Vanini that we find the typical representatives of the humanist attitude to religion, but in Erasmus and More and the Christian Platonists. The new appreciation of the good of nature and the dignity of man and the rational optimism of the humanist ethos demanded a natural theology to justify them. The humanists saw the world as a rational order which could be explained only as the work of divine Reason, as a work of divine art which shows forth the mind of the divine Artist."[8]

Even the pagan Terence had the root of the matter in mind when he wrote "Homo Sum. Humani nil a me alienum puto".

Thus, the humanities promise much more than a mere living. They promise life, wisdom, a spiritual concept of existence, freedom of intellect and will, and the fruitful development of man's ineradicable religious instinct. And of these interests and studies of man, let us choose the three greatest because they include all the others—literature (especially poetry), history and the arts.

Of poetry, it can be said that when poetry is no longer of interest to man, life has become valueless. For poetry implies a vision into the unseen world. When faith was unclouded among the early generations of mankind poetry flourished. Poetry arises from belief and is strangled when belief fails. For belief in the unseen "releases visions and music and the eternal child in the heart of man."[9] If poetry is dying, it may be because we too are moribund or permanently senile. For minds matured by science, faith is a hard thing to come by unless the skylights of the soul are kept open. To inculcate a love of poetry in the physicians of tomorrow will do much to keep that tomorrow humane. Brutality and poetry can not co-exist.

History brings us the largest reward. For it contains everything except the future, and at strange moments even forecasts what is to come. History is like a procession which brings to our feet the precious cargoes accumulated by our fathers. In its tapestried pageant we see the hosts of those who led their generations. These pass in serried ranks holding in their proffered hands, pen or test tube, palate or chisel, mitre or crown, mace or spear by which they laboured towards their destiny. As the march past continues these all turn their eyes to us for our salute of approval or our frown of disdain. And still the pageant proceeds with the countless millions of our fellow humans who, mute and

8. Christopher Dawson: Religion and Culture, Sheed & Ward, London, 1949.

9. Ludwig Lewisohn: The story of American Literature, The Modern Library, p. 570.

undistinguished, yet played their part on the stage of man's never-ending drama.

To ignore the past is to be unaware of the present. How else can we know our strength and our weakness? For the past of our Western Society has endowed us all with those values which should give us the will to survive: from Judea, the intense passion for righteousness and a clear faith in a moral universe and a Divine Providence; from Greece, a love of freedom in politics, perfection and moderation in art, and rational inquiry into our human situation; from Rome, we have gained our sense of the supremacy of law, the rights of all peoples, and the desire for government by consent.

With this birthright, our Western World has developed a civilization which, in spite of the vicissitudes of war, has given to all the earth the means to enrich life, to a degree far beyond that of any other civilization produced by man. And not the least of its achievements is the incomparable gift of modern medicine. It has been within our grasp to bring in a new world of the Spirit, the Kingdom of God upon earth. If we have failed, it is that we have chosen power rather than freedom, preferred the luxury of things to the ecstacies of the spirit. Only in the pages of history may we find the springs of our successes or the sources of our failure. To cherish what Clio the Muse tells us is ours, is the only real basis of sound citizenship without which all will be lost. A curriculum without a broad survey of history takes weapons from our youth who would defend us and saps at the courage which could preserve us.

And what of the arts—plastic and rhythmic? These have met deep-seated needs of human nature. So it has seemed in the past. Can it be that, now endowed with logic and a trained intellect, we are content to let our emotions atrophy? Can we endure a mere mechanical universe and the contemplation of physical phenomena alone? The truth is we can live in two worlds—the work-day one of our professional life, and the other a transcendental realm always open to us through the arts. By painting and music, we probe depths of experience and great mysteries beyond the reach of all physical explanations. And let it be said of the arts, which can not be said of politics, or even of religion, that they have never added to the totality of suffering which is man's apparent lot on earth. The arts have never been the instruments of tyranny, or the source of cruelty, injustice and slavery. Rather do they emancipate. In Blake's fine phrase, "the arts are powers in man of conversing with Paradise".

So let us then stand upon our rights and those of the students whose academic destiny we control. The increasing burden of scientific training must never stifle the breath of life from the souls of our students. The two fields of education — science and humanism — must find integration. How to combine the accuracy, exhilaration, and technical power of the physical sciences with a breadth of vision and sympathy worthy of humanity—that is the task. To perceive the task is to begin its completion. Successful integration has at times been achieved. Perhaps the best example has been found in our own profession of the art and science of medicine. A long tradition of service provides a proud heritage for its present exponents—service based on scientific technology and tempered by wisdom and human understanding.

At his highest, the physician is both scientist and artist. Of the two, the artist is the greater because while science is discovery, art is creation. The greatest of all arts is the art of life itself. This concept of the artist is the needed formula for education—one who has acquired technique by the discipline of science and wisdom by the study of the humanities. The art and science of medicine, a profession wherein technical skill and compassionate understanding are brought to the bedside in even balance, may point the way for the educationalists in all fields to achieve the desired integration. Even as the true artist is creative, so is the physician whenever he with a complete equipment of material and spiritual resource, establishes with his patient that unique individual relationship which is the most precious of his achievements.

If education can succeed in dignifying all tasks however lowly, by a union of technique and wisdom, by stimulating each worker however humble with the artistic vision of creation, it will be because the balance between science and humanism has been corrected. As one of our colleagues, Dr. Rabelais wrote many generations ago, "Science without conscience is the death of the soul".

Medical Science has ensured us a longer life expectancy. To make this long life profitable and interesting we must dignify the retirement years by offering changing interests for changing needs.

Continuous stimulus for intellectual participation will keep the older person abreast of the trends in this modern age. We must help him to grow in tolerance and understanding of the inter-relationships of the life around him.

If his physical activities must decrease because old age has brought decreasing physical strength, we must help him to recognize that he has earned the right to enjoy a less strenuous way of life and that we still need to have him share with us his mellowed judgment, his wisdom born of experience and contemplation.—Helen Noyes in Geriatrics, *The West Virginia Medical Journal*, **46**: 148, 1950.

THE CANADIAN MEDICAL ASSOCIATION

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EDITORIAL

SPECIAL STATEMENT REGARDING CIVIL DEFENCE HEALTH PLANNING AT FEDERAL LEVEL

THE Department of National Health and Welfare has assumed the responsibility for advising the Civil Defence Co-ordinator, General F. F. Worthington, on health and welfare matters. In this connection the Department will initiate and co-ordinate civil defence health and welfare plans at federal levels.

The Department has taken certain preparatory steps such as special training of personnel and collecting basic information on the subject. A co-ordinating committee has been set up, consisting of the following members: Dr. G. D. W. Cameron, Dr. G. F. Davidson, Dr. H. A. Ansley, Mr. R. B. Curry, Dr. K. C. Charron. In addition, a health planning group is being established, with personnel working on a full-time and part-time basis. This group will assist in developing a general pattern which may serve as a guide for provinces and municipalities concerned with civil defence health service planning, and will be under the supervision of Dr. K. C. Charron.

The national professional associations and voluntary agencies interested in various aspects of the program are being invited to co-operate and participate in this planning effort. Similarly, various specialist services within the Department of National Health and Welfare and other Federal agencies will be fully utilized.

Small working parties will be set up to explore various aspects of civil defence health services such as—

1. Civil defence casualty services (first aid, ambulance, emergency hospitals, etc.).
2. Environmental sanitation services.
3. Laboratory facilities.
4. Nutritional problems.
5. Industrial medical services.
6. Special health services (such as pædiatric, obstetrical, mental hygiene, dental, nursing, pharmaceutical, and medical services for evacuees and emergency centres).
7. Medical and health supplies for civil defence.
8. Morgue and burial services.
9. Epidemiology, including health statistics.

The working parties will be composed of persons who have special knowledge in the field under consideration, and the members of the party will be provided with basic information before meeting as a group. In addition, a member of the civil defence health planning group of the Department of National Health and Welfare will sit with each working party to assist in integrating the particular problem under discussion with the overall plan. The first working parties should begin this detailed study early in March, and it is hoped that the whole program can be covered fairly rapidly by this method of approach.

ARTIFICIAL RESPIRATION

THE methods of artificial respiration have multiplied so much in recent years that it is useful to have a comparative study of their efficiency such as has been carried out by the Council on Physical Medicine and Rehabilitation of the American Medical Association.[1] It is pointed out that so far each method has been studied separately by different observers and in a few cases only, or by animal experiment. The Council, on the other hand, bases its report on the work of one group of observers that has compared a number of current methods. They avoided using animals or deeply anæsthetized human volunteers since, in the first case the results would be of dubious value, and in the second there would be some risk, even though remote. They chose therefore to use human corpses so recently dead as to be still warm and before rigor mortis had set in. Some tests also, but not many, were carried out on nine living subjects with voluntarily suspended respiration.

It is at once apparent from this report that some methods of artificial respiration produce very little ventilation, for example those of Schafer and of Eve. In order of increasing effectiveness come the hip-lifting method of Emerson, then the Silvester (the oldest), followed by the more recent Nielsen method.

The choice of a method, however, does not depend only on the amount of ventilation it provides. The prime requirements in most cases are simplicity and ease of performance.

In these respects the Schafer method has deservedly won a high place in all first aid manuals. The Eve method, on the other hand, may require some degree of preparation which would be a serious drawback in an emergency. The Silvester method has the disadvantages that in the required supine posture the tongue falls back into the pharynx, and that it soon tires out the operator. The Emerson method too is fatiguing. The Nielsen method involves some risk of injury to the chest wall.

However, it is possible to combine these methods to some extent, especially if two workers are available. The Schafer prone pressure method can be used along with the Nielsen push and pull method, with approximately double the degree of ventilation produced by one only. Still more effective is the combination of the Schafer with the hip-lifting method of Emerson. All these methods are easy to carry out, and all first aid workers should be trained in their use. This report will certainly give them a clear idea of their respective efficiencies. Even if one method produces more ventilation than another, no mistake will be made in applying the Schafer method of resuscitation, for it can be started without delay, is the easiest to perform and can most persistently be carried on. In artificial respiration it is the first few minutes which are so precious, to be followed often by the long persevering effort which may be needed for complete success. Mechanical devices have their place, particularly in hospitals and in organized first aid centres, but even then the best thing at first may well be immediately applied artificial respiration.

1. Gordon, A. S., Raymon, F., Sadove, M. and Ivy, A. C.: *J. A. M. A.*, **144**: 1447, 1950.

EDITORIAL COMMENTS

Please Tell Your Wife

It has been said, with probably some justification, that the doctor's wife is not kept well enough informed of the social aspect of the annual meeting in prospect. In many cases the Journal, in which the publicity regarding the meeting appears, goes to the doctor's office, where it stays, and the lady of the house does not see what is in it. Now, in recent years, our Ladies Committee for each Convention has taken particular pains to publish welcoming messages in the Journal for the doctor's wives, and to give an idea of the program being arranged for their entertainment. This is being done again this year. The present issue has a message of welcome from Mrs. Church, wife of the President elect, and in later issues there will be details of the various entertainments for the ladies.

It is this material that the ladies committee are anxious to get to the doctors' wives, and we therefore appeal to all our members to pass it on to their home circle, preferably by taking home the Journals containing the notices, so that their wives can see them.

Going to the United Kingdom?

Although Canadian doctors travelling to Great Britain have been able to utilize Canadian funds quite freely, our British colleagues proceeding to this country until recently have been rigidly restricted in the amount of sterling available for their maintenance in the dollar area. Negotiations between the British Medical Association and the Canadian Medical Association for the financing of British doctors temporarily in Canada have now been approved by the Bank of England.

The arrangement is that two British doctors per year will each receive from the Canadian Medical Association the dollar equivalent of £200 on arrival in Canada and a credit in like amount will be established by the B.M.A. to be paid in sterling to the Canadian doctors nominated by the C.M.A.

If you are contemplating a trip to Britain, you can assist this plan by notifying the General Secretary, 135 St. Clair Ave. W., Toronto, and arrangements will be made to receive from you up to $600 in Canadian funds. On your arrival at any port in the United Kingdom a representative of the B.M.A. will meet you and will provide you with the corresponding amount in sterling. Members desiring to participate in this exchange scheme should announce the dates of their visits several weeks in advance to permit all details to be completed.

Affiliated Societies

The Canadian Medical Association welcomes the opportunity of assisting Affiliated Societies which desire to meet in conjunction with the annual meeting. To avoid conflict with the scientific sessions of the 82nd Annual Meeting, the dates of Monday, June 18, and Tuesday, June 19, are recommended as appropriate for this purpose. Executive Officers of Affiliated Societies desiring to arrange for meetings in Montreal during medical week should communicate with the General Secretary, 135 St. Clair Avenue West, Toronto 5, outlining meeting room and other requirements.

MEN and BOOKS

FREDERICK F. TISDALL, M.D., M.B.(Tor.), M.R.C.S.(Eng.), L.R.C.P.(Lond.), F.R.C.P.[C.]*

Alan Brown, M.D., F.R.C.P.[C.], F.R.C.P.(Lond.)

On the occasion of this, the first Frederick F. Tisdall Memorial Lecture, I feel it would be of interest, especially to those who did not have the good fortune to know the late Dr. Tisdall intimately, to give a short sketch of his life, beginning from early infancy. The details of this early period I was extremely fortunate in obtaining from members of his immediate family. A more detailed account of his medical and scientific accomplishments might well provide a subject for a subsequent lecture.

Dr. Tisdall was born in Clinton, Ontario, a fair-sized town in Huron County, on November 3, 1893. He was reported as being a sturdy youngster, and at about six months of age had at least two severe convulsions which undoubtedly would now be diagnosed as tetany. Needless to say, he was energetic and kept everybody on their toes. Another picture of him was that of a small boy trudging home from school, kicking through all the dirty spots on the street and loudly whistling a tune that no one could recognize. His musical ability was limited, but he later learned with considerable effort to play the banjo well enough to earn a place in a High School band. When he was seven or eight years old, he picked up what he thought was a large dud firecracker. Unfortunately it exploded in his face. His brother recalls seeing him on the kitchen table in his father's house with the doctor operating on his eyes, and he reports that something like one hundred pieces of black gun-powder were removed from in and around his eyes. Although his eyesight was damaged to some extent, we can all attest to the fact that he never missed anything.

Between 1907 and 1910 his family moved to Buffalo where he completed his High School work preparatory to entering the University of Toronto. He had little trouble with his examinations and an old report from Buffalo shows that in his final year he had an average of 94.9%. While in Buffalo he became much interested in amateur photography. He followed this up while at the University, and made considerable pocket money by selling pictures and post cards to his classmates.

His boyhood friends and his brother agree that it was always a pleasure, although sometimes strenuous, to have Fred around. He had a personal attractiveness that is rare indeed. After his family left Clinton, he spent his holidays with old friends there, and among other things he helped look after the younger children in the family. This must have been good training for him. For instance, he found that a three year old youngster, with great climbing proclivities, could only be persuaded by a barrage of small stones to come down from a very high hemlock tree that the older ones couldn't climb. It is also stated that the younger members of this family often survived a diet of mud candies that had been made to look almost real. Possibly his interest in nutrition began in this way!

Even at an early age, his business ability was evident. As an example of this, it is reported that as a small boy Fred made sufficient pocket money by watering horses from adjacent farms to rent a rowboat for a very limited length of time. At that time young people did not have everything supplied for them. If they wanted a dance, they worked for it by decorating the hall with bulrushes and Japanese lanterns, and they had to find a sufficient number of musicians to take over, for nothing of course, for the evening. If they wished to have a tennis court they had to make it, and from a virgin hayfield at that! Every year it had to be levelled, hoed, marked and not infrequently it was almost washed out by heavy rains. But that did not matter, Fred could still laugh and do it over again. This is in striking contrast to the ready made amusements that most of the younger generation demand nowadays.

Fred's chief interest in girls at this early age seemed to be in those who were able to supply him with apples. The one most in favour from day to day was the one who could produce the biggest and ripest apples. He had a passion for them and he knew all the orchards and their owners within a radius of five miles. It was reported that one night he took a six quart basket of apples to bed with him. In the morning all that remained were the cores.

While still a boy of school age, he became interested in photography and even at meal time, if he saw the makings of a good picture, he would jump up from the table and go off with his camera. The pictures of sunsets, waves and storms he took when he was about sixteen years old are well worth seeing. As previously mentioned, his photography turned into more than a hobby because he sold some of his work to be made into post cards, and for about twenty years the cards at Bayfield on Lake Huron were made from his pictures.

Later on, when he was at the hospital, his love for animals was evidenced by the fact that his family never knew when he was going to bring home another stray dog or cat from the laboratories. The stray was almost always in poor shape, full of fleas but with a dogged determination to live. Thanks to the combined

* The Frederick F. Tisdall Memorial Lecture, presented at the 27th Annual Meeting of The Canadian Society for the Study of Diseases of Children, at Niagara Falls, Ontario, June 2, 1950.

efforts of the whole household, the animals were nearly always nursed back to health.

Another incident that showed the high regard that his young patients had for him when he was practising pædiatrics, occurred one evening when he and his family sallied forth to a Bay St. theatre. As they entered the lobby, a mother who was herding her numerous offspring around her like a mother hen, caught sight of Fred. Picking up a bewildered three year old she advanced, thrust the child into his face and cried "Kiss the doctor, Jamie, he's the one what saved yer life". Needless to say the commotion caused no little amusement among the theatre patrons and acute embarrassment to Fred Tisdall.

In his earlier years, second only to his love for photography, was his interest in radios and automobiles. His love for cars was an honest family trait, for his father was one of the first automobile owners in Ontario. A soul-shattering incident took place when the family car, driven by his wife, met with an accident. When Fred was called to the phone at the Hospital, his first thought was directed to the car's state. After solicitously asking after the car's health he wanted to know whether or not the family were still in the land of the living.

His first contact with the Hospital for Sick Children was in 1915 when he became House Physician to the infant department of the Hospital. His capacity for getting through a tremendous amount of work was soon evident. In fact he hardly got any sleep, as there were approximately one hundred infants on the ward at the time and there was only one assistant to help him.

In 1916 he went to Baltimore to join a group of young pædiatricians working under John Howland. They were attracted by the opportunity of studying clinical disease by chemical methods. At this time the Harriet Lane was the only American pædiatric department equipped to carry out such chemical studies. According to Dr. James L. Gamble of Harvard Medical School,

> "Fred made a large contribution by his cheeriness and gay sense of humour. Young investigators were not provided with technicians in those days. We made our own measurements and we all worked together in one room. The other members of the group were Marriott, Benjamin Kramer and Graham Ross. In the afternoon Dr. Howland came down and worked with us. None of us I am sure ever had such a good time again. I always think of Fred in that setting. I think of him zestfully stepping about the laboratory and getting more work done per day than any of us. It was the era of the development of micro measurements and the methods which Kramer and Fred worked out for calcium and phosphorus were a great contribution. It completed the armamentarium that enabled Fred, Graham Ross and myself to make a quantitative analysis of the electrolyte metabolism during fasting. The patients were epileptics who were treated by fasting at that time. This was a big job but we were well pleased when we found it disclosed a great deal about body fluid physiology. Even at this early period Fred was an excellent investigator. He planned his work wisely and his interpretation of his results was always sound."

Dr. Edward A. Park, who later became Professor of Pædiatrics at Johns Hopkins, recollects that "Fred lived on Eutaw Place and I lived on Lanvale St. He used to stop at my house every morning about half past eight and we would walk together to the hospital, a distance of about two miles. He would ask me questions about clinical pædiatrics and I would be forced to attempt the answers. He fairly bombarded me with questions. Then suddenly he would destroy my equanimity by pulling a notebook out of his pocket and writing down what I had said. I used to protest but never succeeded in stopping the habit which used to transform our walks from a perfectly informal occasion to one with a certain amount of formality."

In his early life, he showed the same friendly spirit and sense of humour that characterized all his later relationships.

In 1918, through the generosity of Mrs. Walter C. Teagle, sufficient money was given to the Hospital for Sick Children, Toronto, to enable us to set up a small chemical research laboratory. This contribution was augmented the following year by a generous donation, for the same purpose, by the late Sir Joseph Flavelle. Thus began our Pædiatric Research Laboratories. It was soon realized that the accommodation was inadequate for the rapidly expanding research work, so through the generosity and far-sightedness of the Board of Trustees, new laboratories were built in their present location, and were formally opened by the late Sir George Newman, then Minister of Health for Great Britain.

In 1919, we were fortunate in obtaining the services of an outstanding American worker in infant metabolism, namely, Miss Angela M. Courtney, who had previously been an associate fellow in the Rockefeller Institute in New York. She had already made many notable contributions to the study of infant metabolism. This work she continued with us, and during this time she completed thirteen fundamental studies. Upon Miss Courtney's resignation in 1929, Dr. Frederick F. Tisdall succeeded her, and for twenty years he directed our Research Laboratory in a most outstanding manner. His brilliance, enthusiasm, and genius for co-operative organization built up and greatly broadened our effort.

The Hospital for Sick Children has a good reputation in the field of research and many of their findings have proved to be of practical value to adults as well as to children. During the war years we co-operated actively with the Department of Defence, the Department of National Health and Welfare, the Department of Agriculture, the Department of Mines and Resources, and the Wartime Prices and Trade Board on problems of national importance. For over three years more than half the staff of the Research Laboratories worked on these problems at no cost to the Government. Dr. Tisdall played the leading rôle in this work. Dr. Tisdall had an unusual gift for organizing co-operative re-

search, and what was even more remarkable, he was able to guide such projects to fruitful conclusions.

Aside from his outstanding work with Kramer, as a result of which he discovered a new method for the estimation of calcium in the blood, his earliest achievements here, along with two of his colleagues, were related to the establishment of infant feeding on a scientific foundation.

Other projects that were of practical benefit to babies and children, were his studies on the effects of sunlight on rickets. This extensive piece of work received very favourable comment from many parts of the world.

Dr. Tisdall also had a real concern for the welfare of agriculture. This was largely the result of his great interest in human nutrition. He was always ready to help with his services and advice when problems relative to the production and distribution of such products as eggs and milk were presented to him. As the result of his efforts, many agricultural leaders have become conversant with the practical nutritional problems that confront the physician and health worker. In some cases the agriculturalists and the research laboratory worked together on common problems.

His acquisition of a farm a few miles from Toronto, as a hobby, gave him further scope for the development of his ideas in the field of agriculture. He loved beauty in nature and one of the deciding factors in the purchase of this farm was its fine bush, through which ran a meandering stream. He bought hundreds of spring flowering bulbs and planted them in this bush and he took many beautiful and unusual photographs on his farm.

As might well be imagined, a man of his capabilities could not be held within the bounds of a children's hospital, and he rapidly became a world figure. It was in 1937, when he took part in a round table discussion on nutrition at the Milbank Memorial Fund annual conference, that his great natural gifts of leadership and inspiration were especially obvious. Later on, he organized a number of nutritional surveys in isolated areas of Canada. In one study of the conditions of the Indians living around James Bay, their social and economic problems were also investigated. These surveys, which demonstrated the poor physical and nutritional condition of these people, were very valuable in that they demonstrated the need for more health services among the Indians, and recently these services have been greatly expanded.

In 1944 he reported to the Milbank Foundation on the subject of nutrition in England, and soon after this he assisted in organizing nutrition studies in the occupied countries immediately after their liberation.

When the campaign for the new Hospital for Sick Children was contemplated, Dr. Tisdall was chosen by the professional staff of the Hospital to represent them on the Campaign Committee, and great credit is due to him for his efforts in presenting to the public the needs of the Hospital and the past services that it had rendered. The record of the research laboratory did much to persuade the public that the campaign merited their support.

I should like to quote from a letter that I have received from Dr. Boudreau, Executive Director of the Milbank Memorial Fund.

> "Among the many admirable traits of Dr. Tisdall, two were outstanding—first, the energy and vitality with which he threw himself into a project once he saw its possibilities. To him almost nothing was insurmountable. Second, his insistence that the proposed investigations be on problems of practical importance.
>
> "These are but sidelights on the career of a many-sided man. His life had many other facets of which we knew little or nothing. He was an honoured figure among the business and social leaders of Toronto, a distinguished member of his local and national medical associations, a leader among pædiatricians at home and throughout the world. He stood high in government circles, whether in Toronto, Ontario, or Ottawa, and he accepted and honoured many government appointments."

Dr. E. V. McCollum, Emeritus Professor of Biochemistry, Johns Hopkins, wrote me as follows:

> "I believe that Fred was unsurpassed in the most desirable qualities of a man and a pædiatrician—friendliness, intellectual honesty, industry, and enthusiasm about his professional interests. These traits were uniformly sustained through the years.
>
> "Notwithstanding his seriousness as a life long student of medical science, nutrition and biochemistry, he never failed in his personal contacts to bring into the conversation a little sly humour, a good story or a contagious chuckle, especially to reveal scepticism or disbelief on some point under discussion."

Dr. Tisdall served for some years as a member of the Nutrition Committee of the Food and Agricultural organization of the United Nations. This in itself was a signal honour and he proved to be a very valuable member of that committee. Lord Boyd Orr, who was the outstanding first director of F.A.O., was a close friend of Dr. Tisdall and I quote from a letter of his as follows:

> "Dr. Tisdall's abilities as an investigator, a clinician, and organizer are well known. To me the great attraction of Fred Tisdall was his high idealism combined with common sense and a dynamic energy. I never met a man with a finer character and never was in his company without being inspired with renewed zeal for the abolition of hunger and malnutrition. There are few men who have had as profound an influence on my life as Fred Tisdall. The salvation of our threatened civilization depends upon men like him."

The honour which Dr. Tisdall prized most was his Honorary Fellowship in the Royal College of Physicians in London.

Quæ moliebatur perficiamus—"As he began, let us carry on".

I wish to acknowledge the assistance of Dr. Elizabeth Chant Robertson in the preparation of this address.

on call

Published in the Interest of Community Medical Service

Prepared by the Public Relations Committee of the Canadian Medical Association

POTENTIAL ALLIES

Many doctors—many great doctors—have reviled the press. Sir William Osler wrote: "In the life of every successful physician there comes the temptation to toy with the Delilah of the press—daily and otherwise. There are times when she can be courted with satisfaction, but beware! Sooner or later she's sure to play the harlot, and has left many a man shorn of his strength, namely the confidence of his professional brethren." Osler was well acquainted with the difficulties of press relations. On at least one occasion his innocent words were twisted by the press in a way that brought public wrath on his head, yet Osler also knew the value of the press. He often used the columns of the London *Times* to seek support for his many projects.

Osler's paradoxical attitude towards the press illustrates a fundamental principle of medical press relations. The press can be an invaluable ally of medicine, and, unwittingly or deliberately, it can be a dangerous enemy. And the press—included in this phrase is the radio and that growing giant of communication, television—is either of these, an enemy or an ally. There is no neutrality in press relations. The profession is going to have constant relations with the press whether it wants them or not.

Whose Fault is it? The logical answer to the profession's complaints about the press is to try to improve these relations, but to try to improve them on the assumption that the fault lies entirely with the press is not only unfair, but would be fatal to their successful improvement.

In many ways the ideals of the profession and the press are similar. The responsible profession is concerned with the health of the individual. The responsible press is concerned with the health of society. No matter how critical we may be of press performance on specific occasions, no student of human affairs can deny that the responsible press is one of our main safeguards of freedom and, like the medical profession, a leader in the advancement of human affairs. If, at times, the press has been premature in announcing medical discoveries, it has also assisted the profession greatly in spreading of vital health information. If, at times, the press is critical of the profession, it has also been almost unanimous of its criticism of any attempt to limit the freedom of the profession.

Two-way Street. The core of the problem is simple. The press needs the goodwill of the doctors because health is news, important news, and news is the lifeblood of the press. The profession needs the goodwill of the press because the press helps to form public opinion and now, as never before, the profession needs favourable public opinion. As well as in seeking public goodwill, the profession needs the press to help educate the public in health matters, to help raise money for worthy medical causes, and to act as a medium of communication in many other medical fields.

Much of our present difficulty in press relations is based on misunderstanding, a misunderstanding by the press and the profession of each other's problems. Many American state and county medical societies have taken a long step to clear up these misunderstandings in the last two years.

This is the way they have done it.

First, the society has invited leaders of the press to meet with leaders of the profession at an informal dinner. At this dinner discussion of the problems of medical-press relations is invited and, even though this discussion may become heated, the mere fact that the two groups can state their difficulties goes a long way towards clearing them up. Second, out of these informal meetings have arisen, in many cases, formal "Codes of Co-operation" between physicians and hospitals, and the press. Subscribed to by both groups, these codes serve as a guide to all future relations between the press and the profession. They do not usually lay down a rigid set of rules because that would be difficult in such a complicated field as press relations but they do outline certain principles, and they set out the responsibilities of each group to the other.

Typical Code. A typical code, developed in Georgia, has as its purpose "to promote agreement and understanding of mutual problems faced by the medical profession and the press".

Like most other codes, the Georgia code emphasizes the necessity of medical societies and hospitals appointing spokesmen who will be available at all times to the press. "Officers, chairmen of committees, or designated spokesmen of the hospital or medical societies may be quoted by name if it is necessary to authenticate the information given. The press and radio shall have supplied to them a list of spokesmen upon whom they can call for authentic information."

The code outlines the obligations of both groups to the patient. "The physician at all

times will provide the patient with security from exploitation or embarrassment. Names of patients shall be mentioned only with the expressed written permission of that patient, except in cases of disaster."

Under the heading of "Obligations of Physicians to the Press", the code says "physicians or surgeons in private practice shall give as much information as possible provided that it does not jeopardize doctor-patient relationship or violate the confidence, privacy, or legal rights of the patient. Unless it be essential to the story, the physician shall remain anonymous."

Press Obligations. Among the obligations of the press, according to the code, are recognition "that the prime purpose of the physician is to safeguard the life and health of the patient, and they shall refrain from any actions and demands which might interfere with this purpose. Representatives of the press shall execute careful judgment in order to avoid publishing material designated to exploit patient, doctor, or hospital. The press should make reasonable effort to check the authenticity of all medical news and information before publication. This can readily be done by conferring with duly appointed representatives of the medical society before proceeding with publication."

Some American codes are more detailed but, no matter what form the code takes reports indicate that they are working well and that the press relations of medical societies which have established them, have improved greatly.

Some aspects of medical-press relations are covered in the Code of Ethics of the Canadian Medical Association under the headings Advertising, Communications to the Laity, and Radio Broadcasting.

Some of the more important clauses in the Code are:

"The word 'advertising' in relation to the medical profession must be taken in its broadest sense. It includes all those methods by which a practising physician is made known to the public, either by himself or by others without his objection, in a manner which can be fairly regarded as having for its purpose the obtaining of patients or the promotion in other ways of the physician's individual professional advantage."

"Physicians should be extremely cautious in dealing with the Press. A physician should insist, wherever possible, on seeing a proof of what is to be printed under his name or on his authority.

"All opinions on medical subjects which are communicated to the laity by any medium, whether it be a public meeting, the lay press, or radio, should be presented as from some organized and recognized medical society or association, and not from an individual physician. Such opinions should represent what is the generally accepted opinion of the medical profession.

"Discussion in the lay press on disputed points of pathology or treatment should be avoided by physicians; such issues find their appropriate opportunity in the professional societies and the medical journals. (British Medical Association's Decisions.)

"The practice of medical practitioners taking charge of columns in which answers to correspondents on medical questions are printed, is highly detrimental to the public interest and most improper from a professional point of view. (British Medical Association's Decisions.)

"A physician acting in a public capacity, *e.g.*, a Health Officer, may wish to issue to the public warnings or notices regarding public health matters under his own name."

"It is legitimate and even desirable that topics relating both to medical science and policy and to public health and welfare should be discussed by physicians who can speak with authority on the question at issue. In any medium of discussion, but especially in radio broadcasting because of its vast range, it is essential that the physician who takes part should avoid methods which tend to his personal professional advantage. A physician serving in a public capacity is in a different position but even he should see to it that it is his office, rather than himself, that is exalted."

A newspaper code is not in any sense intended to substitute for the existing Code of Ethics. Rather, it helps newspaper men be more conscious of the profession's ethics, provides physicians with more detailed information on correct press relations.

The Canadian Medical Association would be happy to supply any Division or County Association with additional information about these codes and, should it be desired, assist in working out a code that would apply to local conditions.

Working together, under mutually agreeable principles, the profession and the press can become powerful allies for the public good.

Service Club Forum

A doctor who was scheduled to speak on health insurance before the Rotary Club at Welland, Ontario, was unable to fulfill his engagement because of illness. The chairman, for the meeting, also a doctor, substituted a forum discussion at the last minute. Participating in the discussion were the local M.O.H. and three general practitioners. Under the guidance of the chairman the four men discussed the problems of health insurance and how the profession was trying to meet the need for pre-paid medical care coverage.

Reports indicate that the forum held the attention of the audience and stimulated a good deal of interest in the profession's attitude towards health insurance. Other communities in Canada might wish to make use of this forum device. It is recommended that the audience be invited to ask questions at the end of the discussion.

MEDICO-LEGAL

ALLEGED FAILURE TO REMOVE A GALL BLADDER

T. L. Fisher, M.D.*
Ottawa, Ont.

Recently The Canadian Medical Protective Association had occasion to defend one of its members against an unusual charge. It was alleged that although he had told a patient her recent operation was cholecystectomy, in fact he had not removed the gall bladder. The case arose because, after the cholecystectomy in 1948, the patient's condition failed to improve; actually it deteriorated until she consulted another surgeon who operated once again. At operation the second surgeon found a sac containing bile and excised it. When the patient learned of it she assumed this bile-containing sac had been considered a gall bladder by the surgeon and this assumption formed the basis of the action against the first surgeon.

On investigation it was learned the first surgeon was absolutely sure he had removed the gall bladder; that his assistant likewise was positive it had been removed; that both remembered dissecting the gall bladder after operation to verify some suggestion made prior to operation by the radiologist; that the operating room nurse remembered the operation and knew the gall bladder had been removed. Nevertheless, these facts perhaps not being realized by the plaintiff and the plaintiff apparently being clear in her own mind that the second surgeon had dealt with a gall bladder which should have been removed previously, the case was brought to trial. It was begun before a Justice of the Supreme Court of Nova Scotia. The plaintiff's case was very difficult and became much more so when the second surgeon, in the witness box, stated very definitely that he could not identify as a gall bladder the sac containing bile which he had found. In cross examination he stated he had found nothing which would indicate the gall bladder had not been removed previously and there was nothing to indicate poor surgery at the first operation.

When these facts had been established the defendant surgeon's counsel suggested to plaintiff's counsel that the case be dismissed without costs. The Judge then dismissed the jury and an order was taken out dismissing the action without costs to either party.

Recently another surgeon received a threat from a patient in circumstances almost wholly similar. In this case the Association learned more details of the reason for the threat. The second surgeon went into minute detail about his findings, even to the extent of drawing the structures he described, doing it so well in fact that the patient's husband later was able to reproduce the drawings with considerable accuracy. The threat arose then almost certainly because of the injudicious explanations and remarks of the second surgeon after operation. While details are not known about the case which came to court, it is reasonable to assume that a patient would not have brought such a claim against a surgeon unless the second surgeon had been at least injudicious in his remarks.

Cases such as these emphasize the advice the Association gives its members whenever it seems applicable. There seldom, if ever, is any reason for an adverse comment about another doctor's work. Granted a result may be unusual, granted a result may even be poor, a second doctor does not know the full circumstances under which the work was done the first time, and does not know intimate details of the condition. There is every reason why a patient should have a fair, accurate statement of the facts of any case, but such a statement should not carry even an implication that the result was due to poor work on the part of a doctor previously in attendance. Not only do such remarks give rise to needless trouble but sometimes the doctor who made them finds himself in the embarrassing position of being unable to substantiate the imputations he made.

* Secretary-Treasurer, The Canadian Medical Protective Association.

ASSOCIATION NOTES

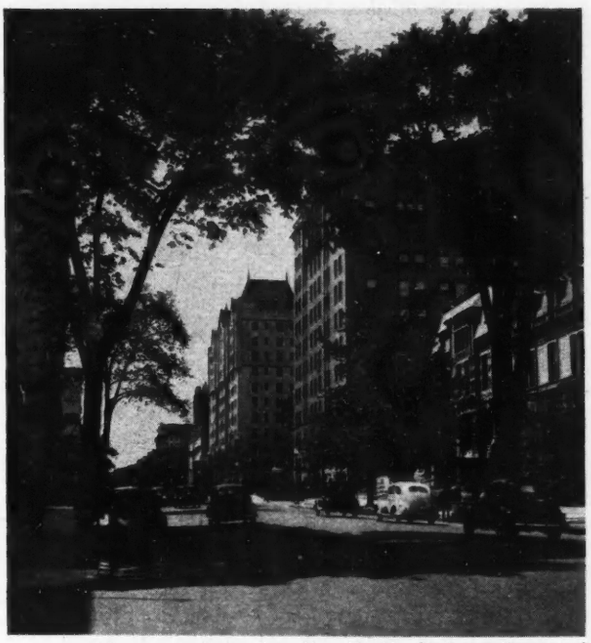
Program Structure at the 82nd Annual Meeting

For our 82nd Annual Meeting to be held in Montreal during the week of June 18, some important innovations have been introduced in the structure of the scientific program. This year for the first time as has already been announced we will utilize colour television as an important means of instruction. The familiar features of round table conferences and general sessions will occupy the mornings of Wednesday, Thursday and Friday, June 20 to 22. Commencing at 9 o'clock each morning, four concurrent round table conferences will be held, and the ever popular general sessions will provide the opportunity for members to hear important contributions from guest speakers from the United States as well as from outstanding Canadians.

Each afternoon commencing at 1 p.m., a continuous program of demonstrations and operations will be projected in colour television. On Wednesday afternoon, the "live" program will consist of a single session devoted to topics of surgical interest directed towards the

general practitioner of medicine. On Thursday afternoon a full program of sectional meetings will be held, while on Friday afternoon the session is devoted to the field of medicine, again directed to the G.P.

The whole scientific program is developing well, and every member will find topics of great practical interest. In the April issue of the Journal further details will be supplied in the form of the preliminary program.



(Courtesy of the Canadian Pacific Railway Company)
Montreal, Que. Sherbrooke Street looking West.

This is Sherbrooke Street in Montreal, on a sunny afternoon in summer. It is not always like this, it must be admitted, and visitors if they have not already experienced traffic jams in Montreal should perhaps be warned that this serene looking scene has a very different appearance most of the time.

But this is not to say there are not more pleasant and easily accessible drives around Montreal. The drive around the Island is one that should be taken. Much of it can be done alongside the river, once the city is left behind. Driving west along the lower Lachine Road one follows the route taken by the fur traders in the early days of Canada, and the windings of the road bear witness to the leisurely progress of those days, and to the probable source of the road as a cowpath. Still, it gives opportunity to see the beauties of the river. The modern road between Lachine and Ste. Annes (at the western end of the Island) runs inland and has no view of the river, but the old Lakeshore road is still used by those in search of beauty rather than quick transport and it is along that road that the tourist should go.

At Ste. Annes the turn to the north and to the east begins, and it is here in the village of Senneville that the drive has the greatest charm. The river and lake are in constant view and the district is all residential, with many large estates and attractive houses.

Then come typical small French villages such as Ste. Genevieve, with many islands lying out in the river, the road winding through woods and farm lands. One of these islands, Ile Bizard, might well be visited for the drive round it, crossing the bridge at Ste. Genevieve.

At Cartierville one is back on a level with Montreal with the width of the Island between. Here one crosses one of the main roads leading up to the Laurentians, a very alluring prospect. But if one keeps on the island the road continues to follow the river and after the temporary depression of passing the large prison at Bordeaux one reaches Sault au Recollet where rapids appear in the river and the same type of winding pleasantly wooded road leads on to Bout de l'Ile where one sees the bridge leading on to the road to Quebec. The return along the south side is less attractive but affords a good view of the mountain.

A longer drive, but one which could be done in about 3 hours from Montreal, is up to Hawkesbury, via Ste. Annes, cross the bridge at Hawkesbury and drive down the north shore to Ste. Eustache. This gives a view of the Ottawa River and the hilly country on the north shore which is unrivalled, and one can always stop at the monks' farm at Oka and sample their famous cheese. At Ste. Eustache one may see the church which still bears the mark of cannon balls on its walls as a memento of the fighting in 1837. The country described is typical French Canada, and few more pleasing drives can be found.

TO THE LADIES

Make Montreal the Mecca

May all roads lead to Montreal—where a Royal welcome awaits you for the June meeting. Montreal has its history. Remember that the convention hotel—The Mount Royal—is built on, or very near the site of old village of Hochelaga. When you are in the hotel close your eyes for a moment or two and let your imagination hold sway; think of this spot as it was four hundred years ago, a few log cabins surrounded by a stockade. The Indians should be only a picturesque memory.

The Ladies Committees have arranged many things for your enjoyment, but—they have thoughtfully left you plenty of time for private sightseeing and shopping. In this old city of Montreal, where French and English dwell side by side in harmony, there are many things to see and do. Try to see Montreal from the "Lookout Point" on top of Mount Royal, or take a trip round the mountain. You will be

amazed at the difference in surroundings and architecture that you see along the way. Then see the Churches and the old Bonsecours Market—these are just a few of the many places of interest. Then there are restaurants of all nationalities within easy reach of the hotels—the ladies at the information desk will tell you how to get to any or all of these.

Then the question—what is the weather like in Montreal after the middle of June?—and what clothes are most comfortable?

Well, it *can* be cool—but more likely to be warm to very warm, at that time of year—so I would suggest a silk suit or dress, and a light wool top coat and perhaps a linen or cotton street dress. Formal dress is usually worn for the Wednesday evening functions.

Come prepared to enjoy yourselves, with others from all parts of this Dominion of ours. We are looking forward to meeting old friends and making new ones. Mary J. M. Church,
Chairman—Ladies Committee

MEDICAL SOCIETIES

Montreal Physiological Society

Montreal Physiological Society, November 27, 1950.

Pharmacology of Injected Cholinesterase.—I. T. Beck, Department of Pharmacology, McGill University.

Bovine enthrocyte cholinesterase (EChE) injected intravenously in rats in doses of 3,000 units/kg. did not produce any change in the tension developed by the gastrocnemius muscle on indirect supra- or sub-maximal stimulation. Rapid close intra-arterial injection of 1-2,000 units of EChE in the cat tibialis anterior preparation was equally ineffective. Addition of 20 units EChE/ml. to the fluid in which a rat phrenic nerve-diaphragm preparation was immersed did not change the response of the preparation to nerve stimulation.

These negative effects may be due to failure of the enzyme to reach the end plate, this, however, seems unlikely in the last experiment. On the other hand, the enzyme may be normally present locally at the endplate in enormous concentration and in excess of that required for efficient acetylcholine breakdown, so that the amounts added may represent only a trivial increase to the amount of enzyme already present. Eccles and coworkers have found that it is necessary to depress the endplate potential to 1/3 normal before conduction fails. It is possible that with the amounts of cholinesterase employed some depression of the endplate potential did occur, but was of insufficient magnitude to reach this critical level.

Factors Affecting the Acetylcholine Found in Excised Rat Brain.—K. A. C. Elliott and Nora Henderson, Montreal Neurological Institute.

The amount of acetylcholine (ACh) found in rat whole brain does not change rapidly after excision, in cortex it falls with time. Less ACh is found in the brains of small than of large animals. Freezing in liquid air causes a considerable loss of ACh. This does not occur with brains from eserinized animals. The ACh. content of the brains of eserinized animals is elevated.

Virtually all the ACh in brain is in the bound form. Acid, mechanical disturbance, freezing, hypotonic suspension medium, and high potassium concentrations accelerate liberation from the bound form. A very low concentration of free ACh may be maintained in brain in the absence of anticholinesterase.

The bound ACh in brain slices increases considerably on incubation aerobically whether anticholinesterase is present or not. This increase is less marked in the presence of a high potassium concentration especially in the absence of anticholinesterase.

Direct evidence was presented that the release of ACh from the bound form is reversible. The possibility was discussed that bound ACh is not necessarily an intermediate in the production of free ACh and that the ACh synthesizing system may be located on the cell surface.

The Effect of the Pressor Hormone of the Pituitary Gland on the Isolated Perfused Rabbit Heart.—F. C. Lu.

It is well-established that the pressor hormone of the pituitary gland causes an intense coronary constriction. It has also been shown by several investigators to augment the contraction of the isolated perfused mammalian heart. The augmentation has been suggested as a result of a direct myocardial stimulation. However, the fact that in the isolated heart the contraction was strikingly augmented when the perfusion was stopped by mechanical means as recently observed in this laboratory (in press) led us to study whether the myocardial effect of this hormone can be regarded as secondary to its coronary effect.

Experiments were carried out in isolated rabbit's heart perfused with Locke solution, using the apparatus described previously (Lu, F. C. and Melville, K. I.: *J. Pharmacol. & Exper. Therap.*, **99**: 277, 1950). Injections of doses of the pressor hormone varying from 0.1 to 10 units were made. After recovery the perfusion pressure was lowered in each case to suitable levels to match the coronary flow as induced by the hormone. The simple reductions in coronary flow induced by the latter method have been found to cause augmentations of heart beat *quantitatively* comparable to those following administrations of the hormone. It is thus concluded that the augmentation of heart beat caused by the pressor hormone is secondary to the coronary constricting action.

Emphasis has been given to the necessity of recording the coronary flow even when the myocardial action of a substance is to be determined in the isolated perfused mammalian heart, as changes in the coronary flow can indirectly affect the heart beat as exemplified in this case.

The Experimental Production of Congenital Anomalies. F. Clarke Fraser.

In the past a number of agents have been found which, when applied to the pregnant mother, lead to the appearance of congenital defects in the young. These include vitamin A-deficient diets, riboflavin-deficient diets, x-radiation, virus infection, trypan blue and anoxia. During a study, conducted by Mr. Theodore Fainstat and myself, of the possible genetic factors influencing the production of such defects, it was suggested by Dr. Hamilton Baxter that we should investigate cortisone as a congenital-defect-producing agent. It was found that large doses (5 to 15 mgm. per day for 3 or 4 days) given in the middle of the gestational period caused abortion or resorption of the embryos. Doses a little lower than this (2.5 or 1.25 mgm. per day for 3 or 4 days) caused a high incidence of cleft palate, and the appearance of a number of other defects in the offspring. The incidence of cleft palate is found to vary with the dosage (2.5 mgm. being the most effective daily dose), the stage (treatment beginning on the 10th day of gestation gives the highest incidence found so far) and the genetic background of the animals used (in two susceptible strains we have found 16 mice with cleft palate out of 38 offspring; in two resistant strains all 24 offspring of treated mice have been normal). The possibility must be investigated that the various environmental agents which, when applied to the pregnant mother, produce congenital defects in the young may act through the adrenal.

La société médicale des hôpitaux universitaires de Québec

Société médicale des hôpitaux universitaires de Québec, le 13 octobre, 1950.

Sub-luxation atlanto-axoidienne d'origine pharyngienne, chez une enfant de 9 ans.—R. Thibodeau.

Nous présentons un cas de subluxation atlanto-axoidienne de cause non traumatique mais secondaire à une infection rhinopharyngée aiguë compliquée d'otite catarrhale. L'évolution de la maladie a été bénigne puisque le seul traitement médical a suffi à guérir ce torticolis: c'est une issue rare. La maladie présente 2 phases distinctes: (1) L'infection première, causale, habituellement une infection de la zone cervicale vg. rhino-pharyngite, otite, mastoidite. (2) La subluxation proprement dite. Le mécanisme de la subluxation serait le suivant: épanchement dans l'articulation amenant une distension de la capsule et des ligaments; conséquemment la subluxation se produit, à la suite d'un mouvement, d'un traumatisme ou d'une manipulation sur la région cervicale.

Les principales manifestations sont la rigidité de la nuque, la déformation et la douleur au mouvement; les autres symptômes dépendent de la variété de subluxation. Le diagnostic de l'affection n'est pas difficile s'il s'appuie sur la radiologie (technique spéciale). Le pronostic dépend d'un diagnostic précoce et d'un traitement adéquat. Si la subluxation ne se réduit pas spontanément, il faudra confier le malade à un orthopédiste.

Société médicale des hôpitaux universitaires de Québec le 3 novembre, 1950.

Inertie diaphragmatique et thoracoplastie. — J.-P. Roger, J.-M. Lemieux et M. Beaulieu.

Deux facteurs principaux peuvent troubler la mobilité diaphragmatique chez le tuberculeux: la pachypleurite locale et la paralysie partielle ou complète du phrénique qui dans un but thérapeutique a été antérieurement écrasé, alcoolisé ou sectionné. L'inertie diaphragmatique favorise les phénomènes atélectasiques homolatéraux dans les suites opératoires immédiates des thoracoplasties. Ces complications pourront être évitées si les interventions sur le phrénique sont réservées à des indications opératoires bien choisies et si à la suite de la constatation préopératoire d'une déficience de la mobilité diaphragmatique, on prend toutes les mesures nécessaires à leur prévention.

On pourra mobiliser fréquemment le patient dans son lit, lui mettre la tête basse, encourager le lever précoce: le tout dans le but de favoriser l'évacuation des sécrétions bronchiques. On ne prescrira que de petites doses de sédatifs capables de calmer les douleurs sans abolir le reflexe de la toux. L'inhalation de carbogène qui est un stimulant respiratoire pourra augmenter l'amplitude des mouvements respiratoires, et ainsi aider à l'expansion des régions en état de sub-atélectasie. Il faudra éviter un pansement trop compressif du thorax surtout à la base thoracique afin de ne pas limiter la ventilation pulmonaire. Enfin, et surtout, dès qu'un patient présente un embarras respiratoire croissant avec une dyspnée de plus en plus importante et une agitation évidente, il faut pratiquer une broncho-aspiration immédiate sans attendre l'apparition des signes classiques de l'atélectasie.

Evolution favorable de formes graves de tuberculose. —R. Desmeules, P. Richard et R. Dion.

Les auteurs rapportent quatre observations qui témoignent des résultats remarquables obtenus par les nouveaux antibiotiques dans le traitement de formes graves de tuberculose. Ils soulignent qu'il y a peu de temps, le diagnosetic de méningite bacillaire, de tuberculose miliaire aiguë, de phtisie galopante, d'entérite tuberculeuse était suivi presque toujours d'un arrêt de mort à plus ou moins brève échéance. Aujourd'hui, de multiples observations prouvent l'action étonnante et souvent inespérée des antibiotiques dans ces manifestations graves de la tuberculose.

Le pronostic immédiat de différentes formes tuberculeuses s'est donc modifié de façon favorable. Le pronostic éloigné de la tuberculose demeure encore une question bien difficile à résoudre. Il est dominé par deux facteurs essentiels: la virulence du bacille et la résistance du terrain. Nous ignorons presque tout de ces deux facteurs. Et tant qu'il en sera ainsi, c'est la clinique qui aura le dernier mot pour apprécier le pronostic éloigné de la tuberculose.

Fistules ganglio-bronchiques au cours de la primo-infection.—Jules Hallé et Lionel Montminy.

Les auteurs rapportent les observations de quatre malades traités pour une primo-infection bacillaire et chez lesquels il a été possible d'observer bronchoscopiquement la rupture d'une bronche par suite de la caséification d'une adénopathie, le passage de l'infection dans la bronche puis dans le poumon, réalisant une épituberculose ou une broncho-pneumonie de Grancher.

La loi de Parrot, énoncée en 1876, constitue encore le fondement de nos connaissances sur les lésions initiales de la tuberculose. D'après son auteur, il n'existe pas d'adénopathie trachéo-bronchique qui n'ait une origine pulmonaire. Pour ce qui concerne la tuberculose en particulier, toutes les fois qu'un ganglion pulmonaire devient le siège d'une infection tuberculeuse, il s'accompagne d'une lésion analogue dans les poumons.

Les observations rapportées tentent à prouver que la loi de Parrot ne semble pas s'appliquer dans tous les cas et que les deux élements du complexe primaire doivent parfois se lire: ganglion d'abord, localisation pulmonaire ensuite. Elles démontrent que c'est à l'examen bronchoscopique de mettre en évidence ou de confirmer l'existence des fistules ganglio-bronchiques. Par ce fait, on a jeté plus de lumière sur la pathogénie très complexe de la tuberculose pulmonaire.

Winnipeg Medical Society

The Winnipeg Medical Society meeting on December 15 was devoted to civilian defence. Ald. J. Gurzon Harvey, chairman of the Civil Defence Committee of Winnipeg and Lt.-Col. Delaney, Manitoba Co-ordinator were present. Lt.-Col. G. L. Morgan-Smith, Medical Officer, Prairie Command, spoke on the medical aspects of civilian defence and Dr. Roy W. Richardson opened the discussion. Two moving pictures were shown, one on the effects of atomic radiation, the other on the organization of medical services.

Dr. M. T. MacFarland, Registrar of the College of Physicians and Surgeons of Manitoba at that meeting denied the allegation that the College was a closed shop for practice in Manitoba. The C. P. & S. is bound, under Provincial Statutes, to issue a licence to any graduate of a Canadian Medical school who presents his basic sciences certificate, his qualifying Medical Council of Canada licence, certificate of a year's internship and $100.00. American graduates of recognized schools may register under the same conditions. Any British subject of an approved medical school, enrolled on the British Medical Register, may register in Manitoba without examination on presenting his credentials including basic sciences and internship certificates. Forty-five doctors have so registered by reciprocity in the past three years. Refugee physicians are subject to the same conditions as graduates of Canadian schools, with the additional safeguard that an Enabling Certificate to write the examinations of the Medical Council of Canada is granted only if the applicant's documents, medical school background and linguistic ability in English or French are satisfactory. Otherwise a year's internship is required.

Dr. MacFarland stated that there is actually no scarcity of doctors in Manitoba. In 1950 there were 775 doctors for 795,000 estimated people, *i.e.*, 1,025 of population per active physician. ROSS MITCHELL

The Physiological Society of the University of Toronto

Dr. J. B. Firstbrook, Banting and Best Department of Medical Research spoke on "The Newer Knowledge of Atherosclerosis", on January 8.

Mr. F. C. Monkhouse, department of physiology, spoke on "The Quantitative Estimation of Blood Heparin Under Various Conditions", on January 15.

Dr. Douglas MacFadyen, professor of biochemistry, University of Illinois, spoke on "A Thermodynamic View of Hormonal Control of Metabolism in Man", on January 22.

Dr. W. J. Linghorne, Banting and Best Department of Medical Research, spoke on "The Effects of Grafts on the Regeneration of Alveolar Bone" on January 29.

LILLIAN A. CHASE.

CANADIAN ARMED FORCES

News of the Medical Services

The Department of National Defence/Department of Veterans' Affairs Medical Co-ordinating Committee recently authorized has held its first meeting. The function of this Committee is to correlate the appropriate activities of the Department of National Defence Medical Services and those of the Department of Veterans' Affairs to ensure that the most efficient use is made of the facilities of both Departments.

Sir Stanton Hicks, Professor of Physiology and Pharmacology, University of Adelaide, Chairman of the Commonwealth Research Nutrition Committee and head of the Catering Corps of the Australian Army spoke to the members of the Inter-Service Medical Committee at a recent meeting.

A standard scale of issue for medical textbooks and periodicals has been approved which will apply to the Royal Canadian Navy, Canadian Army and the Royal Canadian Air Force.

Surgeon Lieutenant Walter M. Little recently transferred from the R.C.N. (Reserve) to the R.C.N. Prior to entering the R.C.N. Dr. Little practised in Goderich, Ontario.

Surgeon Commander W. J. Elliot attended the combined refresher course in otolaryngology and ophthalmology, University of Toronto, January 29 to February 3, 1951.

The following officers were recently appointed to Commissions in the Canadian Army Active Force: Lieutenant J. Lemire, Notre Dame Hospital, Montreal; Second Lieutenant D. A. Cameron, University of Toronto (Faculty of Medicine).

The following officers were recently promoted to the rank of Captain: Captain C. J. Thiel, Hotel Dieu Hospital, Windsor, posted as Regimental Medical Officer, 3rd Battalion, Royal Canadian Regiment. Captain Wm. Crawford, Victoria Hospital, London.

The Advisory Medical Committee to the Royal Canadian Air Force held its quarterly meeting in Ottawa, February 3 and 4, 1951. The meeting was opened by Air Marshall W. A. Curtis, Chief of the Air Staff, Royal Canadian Air Force.

Flight Lieutenant J. McElroy has been posted to the United Kingdom as Medical Officer with 421 Fighter Squadron, R.C.A.F. F/L McElroy is a veteran pilot of World War II.

CORRESPONDENCE

Medical Evidence in Intoxication

To the Editor:

Dr. Donald Mackay's excellent letter regarding medical evidence in intoxication was read with interest, as I have been through a similar harassing and frustrating experience myself recently. The problem of testifying in a drunken-driving charge must confront most practitioners at some time or other, and I know that the local police are discouraged as I am by the number of drunken drivers who get off scot-free.

I have been practising for two years in this semi-rural, "crossroads" community, serving an area of about 4,000 people. Of the two other doctors here, one is semi-retired, and the other makes his home in a neighbouring city. Consequently I am called to a considerable number of highway and other accidents—calls which I cannot refuse—and only too often these end up in summons to appear in court. Offhand I can think of eight medico-legal cases resulting in thirteen different court appearances in the twenty months I have been here.

On starting out, I was given the considered opinion of my chief in regard to examining drunks for the police: "I'd just tell them that in my estimation the man was not drunk, and pretty soon they don't bother calling you any more". Obviously, this is the easy way out, but to me it is not the right way. Not only is a witness sworn to tell the truth, but I feel that a doctor has a moral obligation to come out and say a driver is "drunk" if the diagnosis is certain on reasonable clinical grounds.

Specifically, my case in point concerns a young man whom I was called to see at the scene of an accident seven miles away. On my arrival the driver was almost comatose, probably from a combination of shock and intoxication, though it was impossible to be sure at the time. He was loaded into another car which I escorted thirteen miles further to the hospital. There I was met by a colleague and together we examined the man in detail. In the emergency room there was no question of shock—his blood pressure was 150/90. He was not suffering from any significant injuries. Following his removal to the ward, I observed him in detail for half an hour or so, and found most of the criteria of alcoholism with none of the features of shock.

The preliminary hearing was conducted by the stipendiary magistrate (a layman) with the prosecution in the hands of the local R.C.M.P. constable and defendant represented by counsel. During my 50 minutes on the stand, the defence tried what I have since learned were all the stock tricks of criminal lawyers. He did succeed, I regret to admit, in getting me very angry on the stand—but angry only at his tactics, for he did not make me contradict myself. Being more familiar with court proceedings than our magistrate, he got away with some pretty rough stuff. For instance he remarked that I was "very young and inexperienced",—but the magistrate did reprimand him severely for this. In the end the accused was sentenced to 14 days, but immediately appealed.

My first reaction was to lay a complaint with the bar society, but lawyer friends dissuaded me, saying a lawyer's only ethics are to his client. A short time later I protested to the defence lawyer personally, but his only comments were "it's all a matter of business" and "you were a very biased witness".

Two months later (and three months from the time of the accident) I had to spend an hour and a quarter on the stand in the appeal hearing in County Court. This time there was a full-time judge presiding, and I must say it was a much less formidable experience. However, as in most court cases I have attended as witness, it was impossible for me to get a clear-cut answer to the question of whether I was a plain medical witness or an expert witness. When I left medical school I understood that a medical witness did not have to express opinions, but out here they tell me that a

doctor is in a sort of a special category, and may be asked opinion evidence without being called as an expert witness. Most confusing. In the latter hearing the bulk of my time was spent answering technical medical questions for the judge and prosecution counsel, such as "Doctor, will you tell the court the differences between shock and intoxication from a medical point of view". The judge has not yet handed down a decision, but this delay I can understand owing to the confused state of the law in this matter at the present time. So many decisions have been handed down either way it must be difficult to find a clear precedent.

I heartily agree with your concern for the hopelessness the police must feel in laying these charges, yet my own concern for the moment is how the doctor is supposed to act. Certainly, if the careful examination and honest opinion of two medical men, as in this case, is not sufficient to establish a charge of drunken driving, then I feel that in effect the courts condone this menace on the highways.

The newspapers are frequently reporting this controversy all over Canada. It seems to me that the giving of medical evidence would be a timely topic for the agenda of some of our conventions. Granted, the experts are not agreed on the validity of tests for alcoholism, but I feel that some terms of reference should be laid down for the benefit of medical men who are called to testify only occasionally.

ADAM C. WALDIE
Castlegar, B.C.

Musings of an M.O.H.

To the Editor:

If you are tired of the monotony of reality and wish to fight a make believe war against a make believe enemy either get yourself (or suggest to your Board of Education or School Board) a Woods Light. If you live in a City of over 10,000 population results are guaranteed.

There are four minor communicable diseases mentioned under "Regulations". These are scabies, impetigo, pediculosis and ringworm. Scabies itches, impetigo causes visible running sores, lice bite. If lice didn't bite would they be in regulations? (Note—It is just as sensible to buy a Woods Light as it would be to swab every naso-pharynx. How many diphtheria bacilli and how many pneumococci or meningococci would you find? PLENTY! Each of these is a potential deadly enemy. Don't do it. For if you found them you couldn't do aught about it.)

The use of the Woods Lamp is fascinating. It is best used in a dark room with a pretty, trembling mother accompanying her indifferent offspring. Suddenly you gasp, she gasps. You see little greenish golden glow points like little stars in a June sky. If the case is really good the whole Milky Way is there. Switch on the light. Become brisk, look serious. Give her a sheet of written instructions, (I will send you samples on request) a tube of ointment and a bottle of spray.

If you are really lucky you will find somewhere between 200 and 1,000 cases of epidemic tinea capitis caused by the *Microsporon Audouini* (whoever Audouin was, he started it).

Prepare then to give up all that is real—Immunizations, T.B. Detection, V.D. Control, Well Babies, Prenatal Care. All should be postponed until you have conquered this new enemy. If a mother is foolish and says, "But doctor, look,—I can't see a thing now, Gladys is perfectly well and look at her beautiful curls". —look sympathetic but be stern. Look into the upper right hand corner of the room and intone like an ecclesiastical speaker—"Authorities advise to cut all the hair over the whole scalp ⅛ to ¼ inches. Next please, we are very busy." Order white skull caps until your schools and streets are like meadows with running bobbing mushrooms everywhere. Within a week or so you will be getting phone calls from all over the city. Those new and terrifying types of free citizens, the fixers, the uplifters, will advise you, "Close the shows". "Close the barber shops." "There are some funny looking dogs in our area." The Board of Education will solemnly declare "The situation is serious, something must be done". Your city council will pass a resolution recommending quarantine.

If you are really tops and have the makings of a Napoleon you will make *Life, Time,* and the *Montreal Standard.* Then the real fun will start. A company in Brooklyn will offer to chemically spray fog the whole city free. First line chemical companies will send you free gallons of liquid and pounds of ointment guaranteed to cure in a few days. That amazing and golden tongued perfection of the modern genius of homo sapiens, the drug traveller, will descend upon you in droves and worship you.

All over your city mothers and fathers will spend their evenings pulling dead hairs by the "trial and error method" until all parties exhausted slink off to sleep and dream of glowing hairs. Cheer up! It won't last forever. After the first week cures will start—7, 10, 15, 0, 0, 2, 0. Keep up the battle! Persist! What! A new cure! Dr. Servant in Achigan reported to the Purity Drug Company in Wabos he cured 3 cases in 2 days with a few drops of their new and original product. The Purity Company immediately send you ten gallons, air-express free, prepaid.

All the while the gentle *Microsporon Audouini,* which doesn't understand all this fuss, (after all we are "home" to it) (symbiosis), is going about its usual inoffensive way. When things get too tough it just crawls into its shell. From a week to a few months is his allotted time in any child's scalp. A very few families whose names are honoured have managed to survive until the host reaches puberty. (They are a sour lot anyway and can't stand love. They multiply by fission, like atoms.)

After a while your nurses being sensible about their real job, will grow perfunctory in their examinations. The "Cures" will increase. The last used drug will get the credit—(to be used elsewhere first with no credit). The epidemic will subside. Everybody will be happy and the strange and devious ways of life will resume both for you and for the fungus. Oh well, let's go fishing.

The solution is so simple that no one since Shakespeare's time has even thought of it. Simply call this fungus "Athlete's Scalp" and the other "Ringworm Foot". One is as common as the other and neither means a thing.

J. E. GIMBY
Sault Ste. Marie, Ont.

Silver Nitrate in Cancer

To the Editor:

Has any attempt been made to cause irreversible coagulation of the proteins of cancer cells by silver nitrate? A solution of a strength of one-half of one per cent should be strong enough and will promptly kill any germ or virus that may be present. Owing to its density a tumour grows mainly from the outside and the growing layer needs special attention, treatment of the interior will be simple. The danger of poisoning will be slight. Seventy-five years ago the blue man was a familiar sight in this city. He had had a long course of silver nitrate and oxide was deposited in his skin.

It should be more than worth while to determine the extent to which various chemicals can cause irreversible coagulation in sections of tumours and other tissues and to use the indications as a guide to further investigations. Scar tissue is practically permanent; the writer has several indurations due to experimental injections of one in twenty carbolic acid made some twenty-five years ago. Why cannot a small tumour be converted into scar tissue, its growth stopped by killing the cells, the formation of toxin, whether chemical or organic, prevented and the fatal cachexia inhibited? The simple problem of causing irreversible coagulation and preventing fatal cachexia should not be ignored.

Montreal. RICHARD KERRY

SPECIAL CORRESPONDENCE

The London Letter

(From our own correspondent)

The Passing of Mr. Bevan

It must be confessed that few, if any, tears have been shed at the transfer of Mr. Aneurin Bevan from the Ministry of Health to the Ministry of Labour. His undoubted ability was combined with a personality which was incapable of working with a liberal profession. An inability to respect the other man's point of view, a hypersensitivity to criticism which rivalled that of the most temperamental prima donna, and a gift for invective which could seldom be controlled: all these united to produce a striking and stormy personality, but not the type of man needed to guide the National Health Service through its initial troublesome days.

His successor, Mr. Hilary Marquand, who also comes from South Wales, is a relatively unknown quantity politically. On the other hand, his early days were spent in studying industrial relations, and at one time he was professor of industrial relations at the University of Cardiff. His task has been complicated by the unexpected splitting of the duties of his new Ministry. In the past the Ministry of Health has dealt with housing and local government, as well as with matters of health. With the change of Minister it has been decided to take away from the Ministry of Health all matters dealing with housing and local government and to transfer them to another Government department. Whilst there is much to be said in favour of this move, its sudden announcement must cause considerable interference in the day-to-day work of the Ministry and may cause some delay in the new Minister getting into his stride. It is being hoped that he will bring into his dealings with the medical profession some of the more elementary rules governing successful human relationships which were so strikingly absent in his predecessor's policy. One of the dangers that lie in his path is that he may become too subservient to his permanent officials. To hamstring the medical services with more bureaucracy at this stage would be catastrophic. What is required, and what is still being hoped from the new Minister, is a liberalization of the Service and a slackening of petty regulations.

The Influenza Epidemic

The epidemic which is at present sweeping over the country promises to be the most extensive since 1929. Starting just before Christmas in the Newcastle area it spread rapidly to the Liverpool area and thence to the whole of the north of England. Its spread to the south of the country has occurred at a much slower rate. Although highly infectious, it has not proved to be a particularly serious form of the disease. Most of the deaths have occurred over the age of 55. During the week ending January 13 the increase in deaths in the 126 great towns of England and Wales was almost doubled—from 458 to 890—the largest increase in the last twenty years. The following week the increase was smaller, the number of deaths being 1,099, whilst the number of deaths from pneumonia had actually fallen by 142 to 843. Most of the cases are due to A-prime strain of virus, and the infection seems to have reached this country from Scandinavia. The epidemic has thrown a considerable strain upon the health services and both doctors and pharmacists are having the utmost difficulty in coping with the situation. One of the most disturbing features of the present situation is the difficulty which general practitioners are encountering in getting their acutely ill patients into hospital. It has been reported that in London one out of every three emergency cases was being refused admission to hospital.

Children's Teeth

A further report from Lady Mellanby throws an interesting light upon the incidence of dental decay among children. This report is based upon a comparison of the state of the teeth in 542 children aged five years living in orphanages and institutions, and 560 children of the same age attending independent and private schools in and around London. The standard of comparison was an examination of similar groups made in 1945. Among the children in homes tooth structure had improved and there was a reduction in caries incidence from 10.5 to 7.2%. In the case of children from private schools there was little change in tooth structure and the incidence of caries had only decreased from 14.2 to 12.7%. The average dental structure of the residential-home children was inferior to that of the private-school children, but the difference was less than in 1945. In both groups there was an increase in the amount of dental treatment. In both 1945 and 1950 the percentage of filled teeth among the private-school children greatly exceeded that among the residential-school children. The challenging conclusion is drawn that "few families in Britain, whatever their economic status, provide for their children diets which are as good in their calcifying properties as those served in children's residential homes today, and in few is the intake of cod-liver oil and even milk likely to be nearly so regular or so long maintained."

London, February, 1951. William A. R. Thomson

NEWS ITEMS

Alberta

Dr. E. P. Scarlett of Calgary, an honorary member of the Faculty Club of the University of Alberta, was the speaker at the recent meeting of this Club which now has its rooms in the new Students Union Building on the campus.

Dr. W. B. Parsons of Red Deer is at present convalescing from an operative procedure recently carried out. We are pleased that satisfactory progress is being made. We extend our sympathy to him in the recent loss of a young son who was lost in an accident while at play.

The Crippled Children's Hospital in Calgary is now open and will be a great asset to the southern city. It is one of the most modern institutions of its kind in Canada, very well situated overlooking the city of Calgary.

Dr. Donald Ross of Edmonton is visiting the eastern institutions and organizations relative to the treatment of alcoholics. It is contemplated that a directorship will be created in the province for the treatment of this disease. Dr. Ross is a graduate of the University of Alberta and served in the R.C.N. during the last war.

Dr. Allan Dixon announces the opening of his office in Calgary for the practice of Dermatology. Dr. Dixon holds the degree of M.S. in Dermatology.

Dr. J. J. Porter of Calgary received his Certification by the Royal College in Diagnostic Radiology and Therapeutic Radiology. Dr. Porter was also successful in obtaining his American Board in the same specialty.

Dr. John Ostry formerly of Calgary has opened offices in Edmonton for the practice of urology.

W. C. Whiteside

British Columbia

There is considerable activity at present in connection with shortage of hospital beds in B.C., especially in the lower Mainland area, *i.e.*, Vancouver and environs. The new mayor of Vancouver, Mr. Fred Hume, has interested himself especially in this matter. Shortly after his entrance into office, he made a survey of hospital conditions in Vancouver, and expressed considerable concern over the serious lack of hospital beds. In various addresses, including one to the Vancouver Board of Trade, he has stated that he intends to press for more hospital accommodation as soon as possible.

Also in connection with hospital affairs, the Vancouver Medical Association has urged the establishment of a Metropolitan Hospital Planning Committee, to survey the whole lower mainland area and suggest plans.

The situation with regard to tuberculosis, especially among children, in the Vancouver area is becoming a matter of grave concern, according to two authorities, Dr. Stewart Murray, Chief Medical Officer of the Metropolitan Health Board, and Mr. H. N. MacCorkindale, Superintendent of city schools. Dr. Murray is alarmed by the increase, where for years, owing to the work of the T.B. division of the Provincial Board of Health, and municipal agencies, there has been a steady annual decline. He feels that the shortage of hospital beds has a good deal to do with this. Mr. MacCorkindale finds a rise in the number of new cases among city children of school age.

Port Alberni and Alberni have voted the necessary money by bylaw for the building of the new East Coast Hospital, to cost roughly a million dollars. The B.C. and federal government grants make up a large part of this.

The new Public Health service for the Windermere and Golden districts, is rapidly becoming effective, Mrs. Vernon Marshall, the public health nurse, reports after one year of work. Child Health Clinics have been held at numerous points in the area, and children have been immunized against contagious disease. In all, 1,033 children have been examined in the year by Mrs. Marshall, and 293 by Drs. J. K. Williams of Invermere and Barclay of Golden.

The B.C. total of births for the first nine months of 1950 was 19,537, two less than for the same period of 1949. There were 9,978 boys to 9,559 girls. Infant mortality rate was down from 24.5 per 1,000 live births to 23.1. Maternal deaths were 18 or 0.9 per 1,000 as opposed to 1.2 per 1,000 in the 1949 period.

The General Hospital Board of Salmon Arm has taken an option on five acres of land just outside the city, where it is hoped to build a new 50-bed hospital in the near future.

Postgraduate instruction for nurses is becoming a recognized institution. The Fernie chapter of the B.C. Registered Nurses' Association conducted a two-day school this month. Obstetrics, new hospital equipment, new drugs, and new clinical trends were among the subjects taken up, Miss Fern Trout, R.N., being in charge of the course. In Vancouver similarly a course of the nature is to be put on shortly, and a large attendance is to be expected.

The problem of silicosis in the B.C. Mining industry, appears on the way to being solved. A brief presented to the Royal Commission dealing with the Workmen's Compensation Act, by the Mining Association of B.C., states that it believes that it will be possible within a very short time virtually to eliminate silicosis from the mines of this province. From 1936-39, $5,000,000.00 has been paid by the W.C.B. on account of silicosis. From 1945-50 mining companies have spent more than a million dollars on ventilation, prevention and dust control.

Practically all new cases of silicosis discovered in 1947-49 had been working underground prior to 1936, and most of them prior to 1930. J. H. MACDERMOT

Manitoba

Lieut.-Col. Carl G. Wood, O.B.E., R.C.A.M.C., has been appointed command medical officer, Headquarters Prairie Command. A graduate of the University of Manitoba, he succeeds Lieut.-Col. Morgan Smith who has been selected to attend the Australian Staff College.

Dr. Francis A. L. Mathewson of Winnipeg has been appointed medical director of the Great-West Life Assurance Company effective January 1, successor to Dr. B. H. Olson who has retired.

Dr. J. J. Trudel, St. Boniface, has been reappointed as member of the Board of Governors, University of Manitoba, for term expiring May 31, 1953.

St. Boniface Hospital has installed electroencephalographic equipment. ROSS MITCHELL

New Brunswick

Dr. P. M. Knox, Superintendent of Moncton Tuberculosis Hospital attended a special course in diseases of the chest at Chicago, recently.

Dr. R. B. McKenzie, of Newcastle, was elected a municipal counsellor at the civic election in January.

Dr. G. E. Gauvin, Superintendent of the Sanatorium St. Joseph, at St. Basile, has just completed a postgraduate course in diseases of the chest at New York.

Dr. Sam Milrod, has established an office in Saint John, specializing in surgery.

Dr. H. S. Wright, was re-elected by acclamation Mayor of Fredericton.

Dr. E. A. Petrie, director of the X-ray Department of St. Joseph's Hospital, Saint John, received a serious fracture of his pelvis, plus other bumps and abrasions in a car accident recently. Dr. Petrie was a passenger in a friend's car, which was struck by a skidding car, during a spell of icy road conditions. He will be laid up for a considerable period.

Dr. Ralph P. Myers of Moncton, has been appointed Brigade Surgeon for the New Brunswick Division of St. John Ambulance Brigade.

Dr. W. D. Stevenson, neurosurgeon at Dalhousie University, was the guest speaker at the January meeting of the Saint John Medical Society. His subject was "Acute Head Injuries". A very large audience heard an interesting discussion of methods of diagnosis and treatment of cases of skull trauma. The question and answer period was of special interest.

The municipal council of Saint John has approved the provision of funds to erect a new building for the Provincial Bureau of Laboratories, in connection with the Saint John General Hospital.

Dr. Ronald W. Irving, of Montreal, is now practising in Centreville, where he was located previous to serving in the Royal Canadian Navy.

The staff of St. Joseph's Hospital, Saint John, elected Dr. J. P. McInerney, president of the Medical

Board and Dr. L. Morse chairman of the Standardization Board.

Dr. R. A. H. MacKeen, Director of the Public Health Laboratories of N.B. spoke at the January meeting of the St. Croix Medical Society, on the subject, "The Use and Abuse of Laboratory Methods". He was accompanied on this visit by Dr. Fred Whitehead, secretary of the N.B. Medical Society.

A. S. KIRKLAND

Newfoundland

On January 1 word was received from the Canadian Medical Association that the St. John's General Hospital has now been approved for rotating internships. This program has been worked out in conjunction with the Grace Hospital in St. John's and an affiliation in obstetrics will thereby be provided.

The Corner Brook General Hospital was officially opened on January 5, 1951, by the Premier of Newfoundland. This 100-bed hospital will prove to be a valuable addition to the medical care of the residents of the western section of Newfoundland.

It is anticipated that the new West Coast Sanatorium will be opened in the near future. At the present time the Out-Patient Department is actively functioning. Dr. Alvin Mercer has joined the staff of the Sanatorium as Assistant Superintendent.

Dr. Walter Heneghan who has been practising in the District of St. Mary's for the past year has joined the staff of the St. John's Sanatorium.

Dr. Eric Levy has joined the staff of the Department of Health and is presently resident doctor on the Hospital Ship *Lady Anderson* which is doing duty on the South Coast of Newfoundland.

Dr. J. B. Wilson has opened up practice at Western Bay.

We regret to report that Dr. A. H. Carnell, who for quite a number of years was in active practice in St. John's, died on October 6, 1950 in his 69th year. The medical profession joins with the community at large in expressing sympathy to his family.

CHAS. A. ROBERTS

Nova Scotia

Maritime Medical Care, the profession's health plan in Nova Scotia, was accepted by the Student Body of Dalhousie University at a recent meeting of the Students Council. If ratified by University authorities, and this seems likely, each student will pay 90 cents per month for the eight months of his college year and receive in return complete medical care, less hospitalization and prescriptions.

In November, 1949, *H.M.C.S. Haida* and *H.M.C.S. Magnificent* were the heroes of a daring rescue when they picked from turbulent Atlantic seas eighteen crew members of a United States Air Force B-29. Carrying out his professional duties on this occasion, with one of the small deck craft of the *Magnificent* as his horse and buggy, was Surgeon Captain Eric H. Lee, Atlantic Command Medical Officer. Recently Captain Lee along with Commander Manswick, Captain of the *Haida*, and C.P.O. Roberts, received in recognition of his part in the rescue, which had been acclaimed over the whole of the United States, the Legion of Merit from President Truman.

London has its No. 10 Downing Street, Washington its White House and the little village of New Ross with equally justifiable pride now has its "Doctor's Residence". The six room house is modern in every way and the amount of money for such a community project is not readily come by in New Ross where the wealth of the people lies in greater assets. Into the community pot went the coppers, the dollars and the sweat of a dozen schemes: one hundred days of voluntary labour by forty men, small denomination stocks certificates, a penny a day plan in which each family contributes one cent each day to the fund. Pennies mount fast—in seven years the house will be paid for. It is finished today and President Courtney Keddy has turned over the keys to newly arrived Dr. D. A. Campbell. Of such people are little villages and great nations made.

Dr. D. W. N. Zwicker has been appointed Municipal Health Officer of Chester.

Dr. A. H. Mercer of Halifax and more recently of Lethbridge, Alta., has been appointed pathologist and director of laboratories at Samaritan Hospital, Troy, New York.

From 2 p.m. to 2 a.m. the Executive of the Nova Scotia Medical Society met in semi-annual conclave in Halifax discussing and arguing over the many problems of the doctors' day. Problems on the agenda were Maritime Medical Care and the appointment of a full time secretary for the division, problems which it is hoped will be settled at the general meeting in the autumn.

Twenty-three Nova Scotia hospitals are included in the approval list issued at the year's end by the American College of Surgeons.

Dr. John A. MacDonald (Dal. '45) of New Waterford has been appointed director of the University of New Hampshire's Student Health Service. Under his charge will be the University's Charles Harvey Foot House, an out-patient clinic and hospital and the responsibility for the health of 3,400 students.

All employers in the town of Trenton will now pay one dollar per employee per year to the town for Trenton's share of the new Pictou County hospital to be erected jointly by New Glasglow, Westville, Stellarton, Pictou and Trenton.

Dr. Ian S. Robb of Halifax, United Church of Canada medical missionary, is still serving at a hospital in Chengtuszechuan, China.

From Lithuania via England Dr. Judzs Urbaitis comes to the village of River John, the answer to a long, long search by the Board of Trade for a physician. The third D.P. doctor to set up practice in Nova Scotia's rural areas, Dr. Urbaitis will be warmly welcomed.

Halifax District Trades and Labour Council formally endorsed the use of miniature mass chest radiography after hearing an address by Dr. C. W. J. Beckwith of the Halifax Tuberculosis Hospital.

The Public Health Officers and personnel of the provincial department of Health of the city of Halifax held a three day refresher course at the Nova Scotia Sanatorium at Kentville, under the direction of its medical superintendent, Dr. J. Earle Hiltz. Among those taking part in the program were Dr. V. D. Schaffner, staff surgeon, Kentville; Dr. J. J. Quinlan, assistant superintendent; Dr. D. S. Robb, superintendent, Roseway Hospital; Dr. Hubert Bentley, Sydney, provincial division of psychiatry; Dr. Frank J. Meisner, radiologist, Dr. N. F. McNeil, Dr. J. J. Stanton, Sanatorium staff; Dr. C. J. W. Beckwith, Halifax Tuberculosis Hospital; Dr. D. M. MacRae and Dr. R. C. Young, Halifax; Dr. S. J. Shane, Medical Superintendent Point Edward Hospital, Sydney, Dr. Helen Holden-Quinlan of the Sanatorium staff.

ARTHUR L. MURPHY

Ontario

An Honorary Life Membership in the Hamilton Academy of Medicine was conferred upon Dr. H. B. Van Wyck, Professor Emeritus in Obstetrics and Gynæcology, University of Toronto, at a meeting held on January 17, 1951. In accepting a scroll from Dr. Allan S. Kennedy, President of the Academy, Dr. Van Wyck has become the second living physician to hold this rarely bestowed honour.

The Community Chests Division of the Canadian Welfare Council at a recent meeting in Toronto appointed a committee of five to meet with representatives of national health and welfare organizations to discuss the problems relating to multiple fund raising campaigns. Community chest members have been perturbed for some time at the growing number of fund raising campaigns outside the chests and have felt that these are undermining the "many appeals in one" principle of which the community chests are the foremost exponents.

The chests have called for the creation of a national health and welfare appeals committee with local counterparts all across the country. These committees would study plans for health and welfare fund drives with a view to establishing the validity of their claims for community support. They would examine campaign goals, fund raising methods, internal administration, and purpose for which the money was raised. These committees would be voluntary, without restrictive powers, but with the support of boards of trade, organized labour, service and women's clubs, all racial and religious groups, and health and welfare interests.

A co-operative hospital at Shelburne has been opened by Dr. MacKinnon Phillips, Minister of Health, Ontario. It will accommodate 14 adults and 8 babies. Residents of a large area of North Dufferin donated many hours of work redecorating the building; some of the furniture was made by students at the town high school, canned fruit and vegetables were donated by housewives. Hospital costs were about $2,000 a bed as compared to an average throughout the province of $12,000.

The City of Toronto is planning to spend two million dollars on homes for old people. Dr. George S. Young represented the Academy of Medicine at the meeting of the Welfare Council's Division on Old Age where the plans for the buildings were discussed. There are two types of old people; those who require space only and can look after themselves and those who require care and space. Dr. Young pointed out the need for the establishment of minimum standards for nursing homes; for enforceable requirements for fire and building safety; for a consultative and advisory service to operators of nursing homes; for affiliation with hospitals to make medical services available and to facilitate transfer from one institution to another in case of need.

Lambert Lodge now has 688 residents, 404 up and about, 91 bed cases and 173 senile patients. More than 1,000 applications for admission are pending. Mr. Goodfellow, Minister of Public Welfare, said that he had discussed with the medical faculty of the University of Western Ontario the establishment of a department of geriatrics in connection with the home for the aged which is to be built in London soon.

Dr. Johannes Clemmesen, registrar of the Danish Cancer Registry and secretary of the Expert Committee of Cancer Statistics of the World Health Organization, gave a lecture at the Banting Institute on "The Endemiology of Cancer" under the auspices of the Ontario Cancer Treatment and Research Foundation and the Faculty of Medicine of the University of Toronto.

Sudbury's psychiatric clinic was begun last July. Dr. Tom Dixon, recently resident in psychiatry at Sunnybrook Hospital, is the director; Mr. Dennis Roberts is the psychologist; Mrs. Helen Brown is the social worker and Miss Jeanine Corbeil is secretary and interpreter. This is the pilot clinic and is to be the model for others throughout the province. Dr. Dixon believes that there should be greater facilities for active and early treatment of mental ailments and suggests that psychiatric wards in general hospitals would make early treatment possible.

An adult hearing clinic, aided by a grant of $15,700 from the Atkinson Charitable Foundation, is to be opened at St. Michael's Hospital. The clinic will be directed by Dr. Joseph H. Sullivan. Mr. W. E. Hodges, consulting engineer and research fellow of the department of otolaryngology at the University of Toronto is to be adviser on acoustics and electronics. Miss W. Jones, Reg.N. has been chosen as audiologist.

LILLIAN A. CHASE

Quebec

Appointment of Dr. Jules Mercier, O.B.E., E.D., as senior treatment medical officer and medical superintendent of Queen Mary Veterans' Hospital, and the unification of administration services of the three main veterans' hospitals in the Montreal area has been announced. Dr. Mercier succeeds Dr. C. U. Letourneau.

The three military hospitals—Queen Mary, Ste. Anne de Bellevue and St. Hyacinthe—at present caring for a total of 1,800 ailing ex-service personnel, of which less than 1% are women, were formerly under separate local direction. Under the new plan, each will now come directly under the new senior superintendent, Dr. Mercier, for administration.

At his first press conference in his new post, Dr. Mercier said: "It is our intention to continue to give the best possible medical treatment and service to war veterans as well as to promote the best professional standards with chiefs of other services in this region, and maintain close co-operation with the Department of Veterans' Affairs, and McGill University and University of Montreal."

The announcement also listed the appointment of Dr. E. A. Fergusson, on loan to this region from the Halifax area, as assistant senior treatment medical officer.

Le gouvernement fédéral vient d'affecter plus de $278,000 à l'achat de matériel scientifique et technique à trois sanatoriums de la province de Québec. Les deux premiers sont les sanatoriums du Lac-Edouard et de St-Georges de Mont-Joli, lequel a terminé des travaux d'agrandissement permettant l'installation de 300 nouveaux lits. Le nouveau sanatorium St-Joseph de Rosemont, d'une capacité de 500 lits, reçoit plus de $158,000. Hôpital-école, ce sanatorium est appelé à devenir un centre important de recherches.

Lors de sa dernière assemblée, la Société de Chirurgie de Montréal a procédé à l'élection de son exécutif pour l'année 1951. Président: Dr Charles Lefrançois; vice-président: Dr P. M. Ricard; secrétaire général: Dr A. Couturier; trésorier: Dr Gérard Rolland; archiviste: Dr Guy D'Argencourt; aviseur: Dr Paul Bourgeois.

L'American College of Surgeons a donné son approbation officielle à sept cliniques du cancer siégeant dans les hôpitaux suivants: Montreal General, Division Centrale, Royal Victoria, Jewish General, Hôtel-Dieu, Notre-Dame, St-Luc et Ste-Jeanne d'Arc. L'Institut du Radium a été approuvé comme hôpital pour le cancer.

A sa dernière séance, la Société Médicale de Montréal a procédé à l'élection de son exécutif pour l'année 1951. Président: Dr P. R. Archambault; 1er vice-président: Dr Paul Robert; 2e vice-président: Dr Henri Charbonneau; Trésorier: Dr Paul Dumas; Secré-

taire général: Dr René Rolland; Secrétaire des séances: Dr Pierre Marion; Aviseur: Dr Origène Dufresne.

Le Dr Doma Amyot a été admis à titre exceptionnel au rang de membre associé (fellow) du Collège Royal des médecins du Canada. Le Dr Edouard Desjardins a été élu "Fellow" de l'American College of Surgeons.

Nouvel exécutif de la Société d'Obstétrique: Dr Donatien Marion, président; Dr Pacifique Gauthier, vice-président; Dr Jacques Fortier, secrétaire; Dr Julien Tétrault, trésorier. Le Dr Hector Sanche a été élu président honoraire.

Saskatchewan

The College of Physicians and Surgeons of Saskatchewan entered into negotiations with the Province of Saskatchewan to continue the plan for medical services to the Old Age Pension group of welfare people for another year. The Government is to continue the bookkeeping and to send out monthly cheques but the authority to pay accounts and general management of the fund is to come more directly under the Council of the College through the Assessment Board.

Certain discussions were held with a view to collaboration for radio programs involving health education and public health and possible changes in legislation affecting health affairs were discussed.

The first meeting of the new Council was held in Saskatoon on Saturday, January 20. Dr. J. E. McGillivray was elected President, Dr. H. Gordon Young, 1st Vice-President and Dr. F. D. Sutherland, 2nd Vice-President.

This meeting was primarily organizational but the ground work was laid to continue study on the treatment of Cancer in the Province and Council directed that the methods of supplying prepaid medical care should come under a critical review by the Central Health Services Committee of the College.

Dr. H. C. Burroughs, Lumsden; Dr. G. W. Hutchison, Southey; Dr. M. H. McDonald, Weyburn; Dr. E. E. Kells, Regina and Dr. F. B. Dawson of Maple Creek were elected Senior Life Members having served for forty years in good standing.

Dr. Mary Anna Nicholson has sold her practice in Saskatoon to Dr. Ruth B. Albright and has moved to Bridgetown, Barbados, British West Indies where she will make her home.

The doctors of Saskatchewan are particularly pleased that the ability of their member, Dr. E. A. McCusker, M.P., has been recognized through his appointment as parliamentary assistant to the Ministry of National Health and Welfare.

The doctors of Saskatoon have made personal contributions to the Medical Library at the University of Saskatchewan to the extent of $1,220, and have pledged a further $1,500.

A number of lakes in Saskatchewan have been named after doctors who treated settlers and Indians during the early days of the province in the best traditions of their profession. These names include Charlton Lake, named after Dr. G. A. Charlton, who was in charge of the bacteriological laboratory prior to the formation of the province in 1905; Edwards Lake, named after Dr. O. C. Edwards, of Qu'Appelle Station who was one of the earliest doctors in the province, having practiced prior to 1890; Jukes Lake, after Dr. A. L. Jukes early medical officer of the R.C.M.P., who looked after the health and comfort of the settlers as well as the police.

Also adopted was a placename honouring Sgt. Thomas Dunbar Dakin, son of Dr. and Mrs. Warrin Dakin of Regina, who gave his life in defence of the country during the last war. G. GORDON FERGUSON

ABSTRACTS FROM CURRENT LITERATURE

Medicine

Insulin Resistance Associated with Carcinoma of the Pancreas. Davidson, J. K. and Eddleman, E. E.: *Arch. Int. Med.*, **86:** 727, 1950.

The term insulin resistance does not include diabetic acidosis and cases with acute infections, but is reserved for cases which show absolute refractoriness to insulin which persists more than 24 to 48 hours. The cases reported was admitted on three separate occasions, the last time in a very deep coma which, in spite of every possible device proved fatal. The fact that carcinoma of the pancreas was found at autopsy suggests that the possibility of such a tumour being present should always be kept in mind when one meets such a case.

Of the number of cases reported as belonging to this group, the authors exclude several where there were coexisting conditions which could affect the response to insulin. Poor absorption from the injection site due to insulin lipodystrophy necessitates increased amounts; also there is poor absorption in shock or circulatory failure. A new site with normal subcutaneous tissue, and the use of the intravenous route would simplify these problems.

Pheochromocytoma tumours also cause increased demand for insulin because of the excess of blood sugar derived from hepatic glycogen. Any "crisis" will increase this demand. Insulin allergy is referred to, which causes the question of a circulating antibody or even antibodies fixed in the tissues. P. M. MACDONNELL

Experiences with Dicoumarol in Acute Myocardial Infarction. Bresnick, E., Selverstone, L. A., Rapoport, B., Cheskey, K., Hultgren, H. N. and Sise, H. S.: *New England J. Med.*, **243:** 806, 1950.

Numerous reports during the past few years have attested to the value of dicoumarol in the treatment of myocardial infarction. The majority of these reports have concerned the careful use of the drug under research conditions. To determine its value under conditions existing in a busy general hospital alternate cases at the Boston City Hospital were given the drug during the years 1946 to 1948, 122 cases being so treated and 128 serving as controls.

The results obtained in this study failed to show that dicoumarol caused any significant improvement in prognosis, probably because of the generally inadequate control of prothrombin levels resulting from inexperience on the part of medical personnel, variability of response of patients and laboratory errors in prothrombin estimations. Two deaths resulted from hypoprothrombinæmia. Well controlled dicoumarol therapy was shown to reduce the incidence of thromboembolic manifestations. NORMAN S. SKINNER

An Evaluation of Aureomycin Therapy in Primary Atypical Pneumonia. Schoenbach, E. B., Sweed, A., Tepper, B. and Bryer, M. S.: *New England J. Med.*, **243:** 799, 1950.

Twenty-two consecutive hospitalized patients ill with primary atypical pneumonia during 1946-47 serve the authors as a control group in determining the efficiency of aureomycin therapy in a group of 33 consecutive patients treated during 1948-49. The two groups were comparable as regards sex, age, duration of disease prior to admissions, temperature on admission, degree of pulmonary involvement and proportion developing cold agglutinins. The average duration of fever after admission was 6.4 plus-minus 0.85 days for the control group and 3.1 plus-minus 0.07 days for the aureomycin-treated patients. The average duration of fever after starting aureomycin was 1.8 plus-minus 0.23 days.

The aureomycin caused no significant toxic reactions, although 33% of patients receiving it complained of nausea, 12.8% vomited and 12.8% experienced mild diarrhœa. NORMAN S. SKINNER

The Importance of Recognizing Post-Infarctional Shoulder-Hand Syndrome. Chitwood, W. R.: *New England J. Med.*, **243:** 813, 1950.

The so-called shoulder-hand syndrome which follows myocardial infarction, may be divided into three stages; Stage I, which consists of a painful shoulder, followed or accompanied by painful, swollen and stiff fingers (usually with colour changes varying from a dusky pink to cyanosis); Stage II, which usually brings some relief of the shoulder pain with a subsidence of the swelling in the hands (however, the stiffness and flexion deformities of the fingers increase, and atrophy of the subcutaneous tissues and intrinsic muscles of the hands occurs); and Stage III, which is characterized by progression of the atrophic changes until a Dupuytren contracture results.

The theory of causation of the shoulder-hand syndrome is that it follows decreased local nutrition resulting from decreased cardiac output, hypotension, forward failure with ischæmia and tissue anoxia which act as an active stimulus to the internuncial pool of the spinal cord setting up vaso-spastic responses.

It is important to recognize the persistent pain of the shoulder-hand syndrome and to prevent its confusion with the pain of myocardial origin. Such confusion may result in prolonged and unnecessary bed rest following myocardial infarction, which not only has a deleterious effect upon the shoulder-hand syndrome itself but is likely to give rise to a psychologically crippling cardiac neurosis. NORMAN S. SKINNER

Action of Atropine on the Cardiovascular System in Normal Persons. Nalefski, L. A. and Brown, C. F. G.: *Arch. Int. Med.*, **86:** 898, 1950.

The majority of attacks of angina pectoris occur during the fourth, fifth and sixth decades, the cause of pain being that the blood supply to the heart muscle is inadequate for its needs at the moment of the seizure. Part of this "shortage of supply" is due to anatomic changes in the blood vessels due to arteriosclerosis, but there can also be failure to respond to current needs even where there has been no deterioration in the tissue. A vasoconstricting action may be responsible, such as an increase in vagal tone due to age, in the absence of pathological changes. There is some variance in opinion as to how the vagal tone varies with the years.

The effect of atropine sulphate was observed in 133 subjects who could all be regarded as essentially normal. The objective was to find out what dose was needed to paralyze the vagus, the drug being given i.v. and at basal conditions. Heart rate was reduced for 15 to 20 seconds after i.v. administration, and for 10 to 15 minutes when given subcutaneously. The greatest change in heart rate was found in the people in the first three decades, a much smaller change in the 5th, 6th and 7th. The sturdy thick set persons were more resistant to the drug than the tall thin, asthenic group of patients. Blood pressure reactions to atropine were more brisk in ages 20 to 25 years, before and after those ages patients were more stable in this respect.

Apparently atropine increases the flow of blood in the left coronary artery very greatly. It also increases the cardiac minute volume.

All of which suggests that atropine might be useful in coronary accident. P. M. MACDONNELL

Surgery

The Incidence of Malignancy in Gastric Ulcers Believed Preoperatively to be Benign. Lambert, E. G., Waugh, J. M. and Dockerty, M. B.: *Surg., Gynec. & Obst.*, **91:** 673, 1950.

A group of consecutive cases of carcinoma of the stomach which were supposed to be cases of benign gastric ulcer before operation were examined. In 5 years 550 patients diagnosed benign ulcer were operated upon at the Mayo Clinic and 73 (13%) of them proved to be malignant lesions. The age varied from 25 to 73 years. The duration of symptoms averaged 38 months. Of the group 28% had had rather typical ulcer dyspepsia for from 5 to 40 years. More than a quarter of the patients had noted hæmatemesis or tarry stools; 22% had an associated duodenal ulcer. In 65% the quantity of free hydrochloric acid was within normal limits. In the 9 cases in which gastroscopic examinations were done, the findings were misleading, including evidence of healing under medical treatment.

The conclusion is reached that there is a group of cases of malignant disease of the stomach for which there is no method of diagnosis short of pathologic examination of the tissues removed. The resectability rate was 82% and slightly less than half lived 5 or more years. Temporizing with an ulcerating lesion of the stomach introduces a serious risk. BURNS PLEWES

Surgical Scrubbing with Phisoderm G-11 as Applied to a Maternity Hospital. Reid, E. R., Walter, C. W. and Buck, A. S.: *Surg., Gynec. & Obst.*, **91:** 537, 1950.

As compared with the surgical scrub with tincture of green soap, frequent shorter scrubbings with phisoderm with 3% herachlorophene resulted in a striking reduction in the number of bacteria obtained from hands. Frequent exposure to the agent showed greater reductions in bacterial counts because a persisting protective film is maintained. The short 90 second scrub is effective if it is frequent. This agent renders the hands surgically clean if used properly and frequently.

BURNS PLEWES

Hypothermia—its Possible Rôle in Cardiac Surgery. Bigelow, W. G., Lindsay, W. K. and Greenwood, W. F.: *Ann. Surg.*, **132:** 849, 1950.

The factors governing the survival of dogs at low body temperatures were investigated. The animals were anæsthetized, cooled by using a blanket with circulating alcohol and warmed in a warm water bath. The state of lowered body temperature and oxygen requirements might permit operations on the "bloodless heart" and perhaps transplantation of organs. The dogs could safely be cooled to 20° C. There was a marked fall in cardiac rate, cardiac output and blood pressure as cooling progressed and a rise on re-warming. Vasoconstriction was observed. Cooling also caused a rise in venous pressure and this was a valuable guide to the condition of the heart, for if it persisted it was followed by "cardiac crisis" which could be temporarily forestalled by venesection. Ventricular fibrillation usually caused death between 16 and 22° C. Observations of the effects of several anæsthetic agents, electrocardiographic changes, the appearance of the fundi oculi, cardiac stimulation and massage, etc., are recorded and discussed. BURNS PLEWES

Effect of Analgesics and Antispasmodics on Common Duct Pressures. Curreri, A. R. and Gale, J. W.: *Ann. Surg.*, **132:** 348, 1950.

Using patients with cholecystostomy tubes and a novel method of recording changes in the fluid level in a manometer, studies were made of changes in pressure in the common duct. Several different drugs were used in one patient on different days to insure proper comparison.

Activities such as talking, walking, coughing and straining increased the common duct pressure. The pressure required to overcome the tone of the sphincter of Oddi causes neither pain nor discomfort, but analgesics and parasympathetic stimulators (*e.g.*, urecholine) may increase sphincter tone so that pressures rise to a pain-producing level at 20-plus cm. of water. Analgesics are powerful sphincter stimulators and may accentuate rather than lessen biliary colic so that an effective dose must produce marked central depression. Antispasmodics were disappointing except amyl nitrite and aminophyllin. Amyl nitrite was most effective in lowering common duct pressure but its effect is short-lived. Large doses of nitroglycerine and papa-

verine were also effective, but even 1/33 gr. of atropine failed to alter ductal pressure appreciably. Synthetics such as syntropan, pavatrine, amethone, etc., have no apparent effect on the sphincter, and neither did fatty meals, nitranitol and etamon. Splanchnic block does control pain due to increased common duct pressure. BURNS PLEWES

The Surgical Treatment of Hyperparathyroidism. Reinhoff, W. F.: *Ann. Surg.*, **131:** 917, 1950.

Since the first operation 25 years ago by Mandl and the isolation of parathormone by Collip and Hanson, the determination of hypercalcæmia, hypophosphatæmia, hypercalcuria and hyperphosphaturia renders the diagnosis of hyperparathyroidism a certainty. If the cerebrospinal fluid calcium is over 6 mgm. % it is evidence against hyperparathyroidism. Sometimes the site of the parathyroid adenoma is difficult to ascertain and the differentiation of adenoma from diffuse hyperplasia may complicate the operation.

The number of cases reported since 1903 is brought to 597 including the 27 cases on which this paper is based. Renal lithiasis is the most common complication of hyperparathyroidism and it is suggested that the calcium phosphorus ratio be determined in all cases of renal stone, for this is a more common early sign than decalcification fibrosis of bone. In 25 out of the 27 cases adenomata were found and of these 2 had 2 adenomata, 1 had carcinoma of the parathyroid, 1 had diffuse hyperplasia of 4 parathyroid glands. Postoperative tetany did not occur in patients with renal signs, but in those with extensive skeletal changes it occurred 6 times. Metastatic calcification was found in 6 of the 27 patients. There was 1 postoperative death from tetany, and follow-up showed that 9 died in from 3 to 11 years of hypertension though apparently cured of hyperparathyroidism. Of the 14 patients with skeletal complications, one had complete reossification within a year. Renal damage may progress postoperatively. Emphasis is on the main goal of making the diagnosis before obvious and serious complications have occurred.

The anatomy and embryology of the parathyroid glands are described and the sites at which adenomata may be found are illustrated. At operation all normal parathyroid glands should be carefully preserved as should thymic cervical rests. Parathyroid adenomata are spherical and average 7 gm. in weight, varying from 0.4 gm. to 120 gm. and are dark blue in areolar tissue and golden brown on section.

It is suggested that a predominance of clear Wasserheller cells occurs in those tumours associated with renal complications, and a preponderance of chief cells in those cases with skeletal changes. It may be that the goitrogenic thiouracil drugs stimulate the formation of parathyroid adenoma when administered over a long period of time. BURNS PLEWES

Obstetrics and Gynæcology

Breast Abscess. Newton, M. and Newton, N. R.: *Surg., Gynec. & Obst.*, **91:** 651, 1950.

The development of a postpartum breast abscess may be best understood if it is regarded as a final stage in the failure of lactation. Three factors are important (1) trauma, (2) infection, (3) stasis. Emotional stimuli, *e.g.*, pain, emotional upheaval or fear may influence the let-down reflex in human lactation. This reflex is a mechanism, entirely distinct from the secretion of milk, by which the mother actively expels the milk which is already in the breast in response to the stimulation of sucking. Inhibition of the reflex causes stasis in the breast since the milk which has already been secreted is not expelled.

In a recent series in England lactation was continued on the healthy breast while milk was pumped from the affected breast, boiled and then given to the baby. Eighty-eight women were carried through an attack of acute suppurative mastitis in this way without stopping lactation and the babies suffered no ill effects.

Since the incidence of breast abscess is less than 0.1% we are dealing with a relatively small proportion of the parturient population. The problem of prevention is therefore as important as that of cure. It is essential to help mothers avoid discomfort, embarrassment, emotional disturbances and distractions during the puerperium both in the hospital and at home.

ROSS MITCHELL

The Use of Radioactive Cobalt in the Treatment of Carcinoma of the Cervix. Barnes, A. C., Morton, J. L. and Callendine, G. W.: *Am. J. Obst. & Gynec.*, **60:** 1112, 1950.

After activation in the nuclear reactor, cobalt forms a relatively stable isotope with a soft beta ray which requires minimal shielding, and has a homogeneous gamma irradiation. It can be machined to any desired shape, prior to activation, and constitutes an easily handled radioactive material. Advantage has been taken of these qualities in the application of cobalt-60 to the treatment of the forty patients with carcinoma of the cervix here reported. Fine calibre needles have been used which can be fashioned to the desired length and strength, and template guides have been introduced to provide precision of needle placement. These factors tend to increase the safety of multiple source intrapelvic irradiation, and a method is offered whereby an evenly distributed pattern of radiation can be designed to fit the patient's pelvis and the spread of the lesion.

ROSS MITCHELL

The Management of the Pregnant Diabetic Woman and Her Newborn Infant. Reis, R. A., de Costa, E. J. and Allweiss, M. D.: *Am. J. Obst. & Gynec.*, **60:** 1023, 1950.

Careful diabetic management throughout pregnancy and adequate prenatal care without any form of sex hormone therapy, when coupled with individualization in the time and method of the termination of the pregnancy, yield excellent results.

The lethargic, oversized and overweight baby must be treated as if he were a premature baby. Such a baby demands special care. ROSS MITCHELL

Anæsthesia

Lumbar Puncture Headache. Glesne, O. G.: *Anæsthesiology*, **11:** 702, 1950.

It has generally been believed that in the majority of cases headaches following spinal anæsthesia are the result of leakage of spinal fluid through the dural puncture, with subsequent changes in cerebrospinal fluid dynamics, loss of cushioning effect on the brain and resulting pain owing to pressure or traction, or both, on sensitive brain structures and large vessels. This investigation is based upon a study of changes in spinal pressure after lumbar puncture in two groups of patients: 20 patients who developed headache after spinal anæsthesia and a control group of 30 patients who remained free from the complication.

It has been demonstrated that the size of the needle used has a great deal to do with the relative frequency of headache following spinal anæsthesia as it developed in 60% of cases when a 16 gauge needle was used and in 48% when a 22 gauge needle was employed according to Redlich *et al.* In the group of patients which were the subject of the present investigation, spinal fluid pressures were taken on all patients at the time of the initial puncture and again forty-eight hours later. Thirty patients who did not develop headache had this second puncture done as a control and twenty patients who did develop headache were subjected to measurements of the spinal fluid pressure and were treated by the injection of 5% glucose in saline solution. The volume of the latter solution used should not exceed 50 c.c. as the author found that the injection of larger quantities caused sacrococcygeal pain for two to four hours in three patients.

The evidence definitely reveals that the pressure following spinal puncture is lower in the patients who developed headache than in the control group and the etiology of postspinal headache, based on the leakage theory, is substantial. The results of treatment of postspinal headache by the injection of 5% glucose in saline are reported and while this is a small series so that it does not warrant undue enthusiasm, it does indicate its usefulness in the treatment of severe cases with the prospect that symptoms may be ameliorated in at least 50% of the cases. F. ARTHUR H. WILKINSON

Industrial Medicine

Controlling Insect Pests Through Their Nutritional Requirements. *J. Econ. Entomol.*, **43**: 399, 1950.

Recent unbridled chemical warfare against insects has vastly increased the lopsided balance of nature in connection with insects and agriculture. Control of these pests through their diet or their nutritional needs, is suggested in this editorial. Although present-day knowledge of the relation of insect form determination and fecundity to nutrition, is still limited, there are definite implications that striking results in insect control may be effected through their nutritional requirements.

Insects are and have been the most adaptive of all animal life to changing foods and shifting climatic conditions. It has however been observed that shifts in diet may affect not only their well-being but their anatomy and their fecundity. There are many familiar examples in nature and similar observations have been made on test insects. The diet given to the honey-bee when it is a grub determines its future rôle—worker or queen. The proper diet given later may change the sterile adult workers into egg layers. Wing formation in aphids is due to the chemistry of the plant sap which they consume. Chinch bugs and greenhouse thrips are two pests both of which have definitely been shown to be influenced by nitrogen in the diet. For example, on a diet of plants grown in nutrient solutions low in nitrogen, the chinch-bug is normally more vigorous, lives longer and lays more eggs than when on a diet high in nitrogen. By stepping up soil fertility, therefore, both of these pests should be influenced. On the other hand other major pests seem to choose and require crops grown on highly fertile soil. It might be possible to affect these pests unfavourably by shifting the relative levels of the principal soil minerals and the minor elements.

Much of what has been done so far along these lines has been rather exploratory but it seems to indicate that by using modern practices in conserving and rebuilding the fertility of the soils, insect control could be effected. This would be largely by reducing their reproductive potential. MARGARET H. WILTON

Pensions Are Not Enough. Woodhouse, D. L.: *Indust. Welfare and Personnel Management*, **32**: 89, 1950.

That continued occupation for the elderly is desirable in view of the national need, that it is easier in view of changed conditions in industry, and that it is possible as a result of advances in medical science, is claimed by the author of this article. He maintains that the present policy for the ageing population is outmoded. Pensions should not be allowed to continue as a means of rejecting the elderly with a salve to the conscience of society, but rather they should be used to offset reduced earning capacity when this of necessity comes about. Furthermore they must act as an incentive to continue in work even as they make provision for those who cannot.

In a concise statement the author presents his views on the problems of employment and the ageing population. He refers briefly to the methods of dealing with the old, infirm and destitute, as carried out in England since the beginning of the 19th century. In his opinion, conditions under present provisions are not satisfactory, either from an economic point of view or from the point of view of physical and mental welfare. There is urgent need for a reorientation on the part of society, industry and often of the elderly themselves, towards employment. Reference is made to the Birmingham area where some firms have special workshops in which elderly people are working effectively and profitably.

In making suggestions for the future the author emphasizes that success would be dependent on a change in attitude on the part of trade unions, on an assumption of full employment and on international economic activity. He indicates difficulties that would arise and suggests ways in which they could be resolved. Reference is made to the proposition put forward by Mr. M. T. Bloom in 1949, that all firms should have a retirement preparation program as an integral part of their personnel policy. MARGARET H. WILTON

OBITUARIES

Dr. W. T. Barrett died on December 14, 1950, in Victoria, B.C., at the age of 83. Born in Barryfield, Ont., he studied medicine before the turn of the century at the University of Manitoba. Dr. Barrett went to the Yukon with the Guggenheim Mining Co., and served on the staff of St. Mary's Hospital in Dawson City. In 1914 he went to England, where he did postgraduate study. On his return to Canada in 1915, Dr. Barrett set up a practice in Victoria. For many years he was one of the leading physicians on the staff of St. Joseph's Hospital. After his term with St. Joheph's, Dr. Barrett was appointed acting head of the Health Insurance Commission. He retired two years ago. He is survived by his widow, and a son.

Dr. W. Grant Breckenridge, aged 38, chief of orthopædic surgery at Children's Memorial Hospital, died suddenly on December 15, 1950. Born in Peterborough, Ont., he obtained his degree in medicine at Queen's University, Kingston, and interned in hospitals at Ottawa and Boston. Dr. Breckenridge joined the Royal Canadian Navy in 1941 and, after serving in ships in the North Atlantic, headed the R.C.N. hospital at Niobe, Scotland. He joined the Children's Memorial Hospital in 1946 and was chief of orthopædic surgery and also head of the cerebral palsy clinic. He was a fellow of the Royal College of Surgeons. Survivors include his widow, a son and three daughters.

Dr. Arthur Silver Burns, aged 71, died recently in New Jersey. Dr. Burns was born at Kingston Station and following his early education in Kingston schools, he attended Horton Academy, Wolfville, and then entered Acadia University where he graduated with the degree of Bachelor of Arts in 1898. Five years later, he received his medical degree from McGill University. He began general medical practice in Bridgewater in partnership with Dr. H. A. March and in 1905 started his own practice at Bridgetown. Seven years later he came to Kentville. During the First World War he served with the Canadian Army Medical Corps in England and France with the rank of captain. Later he went to New York where he took special studies in psychiatry at Columbia Medical School. Following this he returned to Kentville, but did not engage in active practice except when the need arose through a shortage of doctors. He was a member of the Canadian Medical Association and of the Nova Scotia Medical Society.

Dr. Burns served for a number of years on the Kentville Town Council and was active in community affairs during his residence here which extended over 30 years. He was widely known throughout the Annapolis Valley.

Dr. Waldo Brock Clarke, aged 73, staff member of Vancouver General Hospital for many years, died on December 17, 1950. Dr. Clarke was born near Schomberg, Ont., and received his medical degree from Toronto

NPH INSULIN

Following an extended period of clinical trial there is now generally available a modified Insulin preparation known as NPH Insulin. The product is distributed as a buffered aqueous suspension of a crystalline preparation of Insulin, protamine, and zinc. It is supplied in 10-cc. vials containing either 40 or 80 units per cc.

Crystals formed of Insulin, protamine and zinc in NPH Insulin

NPH Insulin exerts a blood-sugar-lowering effect extending for slightly more than a 24-hour period. In most instances this new preparation has been found to act more quickly than Protamine Zinc Insulin but for a shorter period. Probably because of the fact that NPH Insulin is a suspension of crystals, its use has been found advantageous in cases where it is desired to administer Insulin and a modified form of Insulin in a single injection without appreciable alteration of the effect of either of the two preparations.

University in 1904. He spent two years in postgraduate study in Edinburgh and Glasgow and returned to Canada to set up practice in Whitehorse, Y.T. In 1920 he moved to Saskatchewan and practised there until 1936, then he went to Vancouver. He was in private practice until 1942, when he joined the staff of General Hospital. In 1948 he joined the staff of Royal Columbian Hospital in New Westminster, staying there until ill health which preceded his death forced him to retire. He is survived by two sons.

Dr. John Harper Cormack, native of Kingston, Ont., and graduate of Queen's University, died in Toronto on December 9, 1950. He was 83. He came to Toronto 30 years ago, and established a practice. Earlier he practised in Ottawa and New York as a specialist in ear, eye, nose and throat diseases. He is survived by a son.

Dr. James A. Crozier, aged 75, died at Port Credit, Ont., December 31, 1950. Born near Orangeville, Ont. Dr. Crozier graduated in arts from Queen's University in 1896 and in medicine and surgery from McGill University in 1899. That year he joined the Lord Strathcona's Horse and served in the Boer War. Upon returning to Canada Dr. Crozier practiced medicine at Copper Cliff, Ont., briefly before moving to Port Arthur early in the new century. An active medical practitioner he was medical consultant at Port Arthur for the C.P.R., and the C.N.R. He was a member of the advisory board of the old Railway Marine and General Hospital in Port Arthur and played a prominent part in the establishment of the new General Hospital. He served overseas in the First World War with the 8th Battalion, C.E.F., and at the close of hostilities resumed his affiliation with the L.S.R., and became commanding officer. He is survived by his widow and two daughters.

Dr. J. J. Diner, formerly of Winnipeg, died on January 4 in Vancouver where he had resided for the past three years. He was born in Winnipeg in 1902 and graduated from Manitoba medical college 23 years ago. After graduation he studied in England, subsequently taking up his practice at Spalding, Sask. He then moved to Melfort, Sask., where he resided for five years before moving to Vancouver.

Dr. Warren Doan, aged 83, who had practised medicine continually for 57 years in Harrietsville, Ont., died on December 20, 1950, in London, following an operation. He was born and spent his early years at New Sarum, and after graduating from Trinity College, Toronto, went to Harrietsville and started to practice. That was in the horse and buggy days, and realizing that lives were being lost because local and district communication was too slow, he organized the Harrietsville Telephone Association, and was its president and general manager for 48 years. On the occasion of his 50th year of graduation he was presented with a gold headed cane by the Ontario Medical Association. Surviving is one daughter.

Dr. E. J. Eacrett, long-time physician of Mission City, B.C., died suddenly on December 13, 1950. Dr. Eacrett had been a resident of Mission City since 1926. Previous to that he practiced for many years in Lloydminster, Sask. He is survived by his widow, three sons, and a daughter.

Dr. Charles E. Freeman of Moose Jaw, Sask., died on January 2. Probably the oldest practising physician in the province in length of service, Dr. Freeman began his professional career at Londonderry, Nova Scotia, in 1900. Dr. Freeman was born in Nova Scotia and graduated from Acadia University in Wolfville with a bachelor of arts degree in 1896. He studied medicine at McGill University, graduating in 1900. He went to Moose Jaw in 1908, specializing in eye, ear, nose and throat. He was a member of the Dominion and Saskatchewan medical associations and the Moose Jaw Medical Society.

Surviving are his widow, a daughter and two sons.

Book Reviews

Essays in Surgery. Presented to Dr. W. E. Gallie, on the occasion of his retirement from the chair of surgery in the University of Toronto. 584 pp., illust. $9.50. University of Toronto Press, Toronto, 1950.

This attractive volume, containing forty-three contributions by Dr. Gallie's colleagues and pupils, has dual merit as a tribute of affection and esteem for one of the great surgeons of this era, and at the same time as a compilation of papers of sufficient quality to deserve preservation alongside the imposing list of achievements of this famous chief of a celebrated school.

The fields in which Gallie set his masterful mark are well represented, and the book also fortunately contains articles of importance (Dr. J. L. McDonald's contribution on fractures in the elbow region is a good example) that have escaped the attention they deserve. When new printings of this book are made, it would add to the historical interest if the present title and appointment of each contributor could be added in the list of contents. The book is a moving tribute to a pioneer, and a handsome reward for his work.

The Physiology of Tissues and Organs. D. H. K. Lee, Professor of Physiological Climatology, The Johns Hopkins University, Baltimore, Maryland. 159 pp., illust. $4.75. Charles C. Thomas, Springfield, Illinois; The Ryerson Press, Toronto, 1950.

It is a truism that the start of a new discipline is at the same time a start in a new set of terms and concepts which must be systematized or indexed at an early date. It is unfortunate that this should be so, for students of the discipline become committed to an "index system" which may, as the subject broadens, prove inadequate. In Dr. Lee's estimate this state of affairs exists now in physiology, where our continuation of systematic indexing has completely obscured the general principles common to all tissues and cells. To meet the problem of teaching students he believes that systematic physiology should be preceded by an outline of the broad general truths which apply equally throughout the body. With this background, the student can then see how these principles apply to specific organs and then, finally approach the modifications and special conditions applicable to systems. This is indeed a fresh approach. This little volume presents Dr. Lee's background material for the student of human physiology. It is well done and will save the student hours of tedious memorizing, giving him instead a logical thought framework upon which to build.

Studies on Tumour Formation. G. W. deP. Nicholson, late Professor of Pathology, Guy's Hospital, London. 637 pp., illust. Butterworth & Co. (Publishers) Ltd., London, 1950.

The late Dr. Nicholson was a militant scholar who wrote for an intimate circle. The personal, often reiterative style of his tumour studies which have now been collected and published posthumously, makes it easy to imagine oneself sharing a conversation at his fireside. The studies originally appeared in Guy's Hospital Reports between 1922 and 1938. It was a period when the impetus of advance in pure morbid anatomy had been checked and there was a need for someone to do a little weeding in the field of theory. Nicholson does this weeding effectively and with clarity and his criticisms of Cohnheim's "cell rests", Gravity's theory of the origin of renal tumours and Wilms' views on teratomas are worth reading from a historical point of view as being excellent exercises in inductive reasoning. As Professor Nicholson says in the foreword "Topics which are dealt with more authoritatively . . . than in any other writings include the general structure of tumours, minor malformations, heterotopia of tissues, metaplasia, mixed tumours and the teratomas". The book is laced with curious histological detail which will offer an enduring attraction to the pathologist and is illustrated with informative little drawings made by Nicholson himself.

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Books Received

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Books are acknowledged as received, but in some cases reviews will also be made in later issues.

Stress. Hans Selye, Professor and Director of the Institut de Médicine et de Chirurgie Expérimentales, Université de Montréal. 822 pp. illust. $14.00. Acta Endocrinologica Inc., Montreal, 1950.

A History of English Public Health 1834-1939. W. M. Frazer, Professor of Public Health, University of Liverpool. 498 pp. illust. $6.75. Baillière, Tindall and Cox, London; Macmillan Company of Canada Limited, Toronto, 1950.

Virus and Rickettsial Diseases. S. P. Bedson, Professor of Bacteriology, London Hospital; A. W. Downie, Professor of Bacteriology, University of Liverpool; F. O. MacCallum, Director Virus Laboratory, Ceneral Public Health Laboratory; C. H. Stuart-Harris, Professor of Medicine, University of Sheffield. 382 pp. illust. $4.55. Edward Arnold & Co., London; Macmillan Company of Canada Limited, Toronto, 1950.

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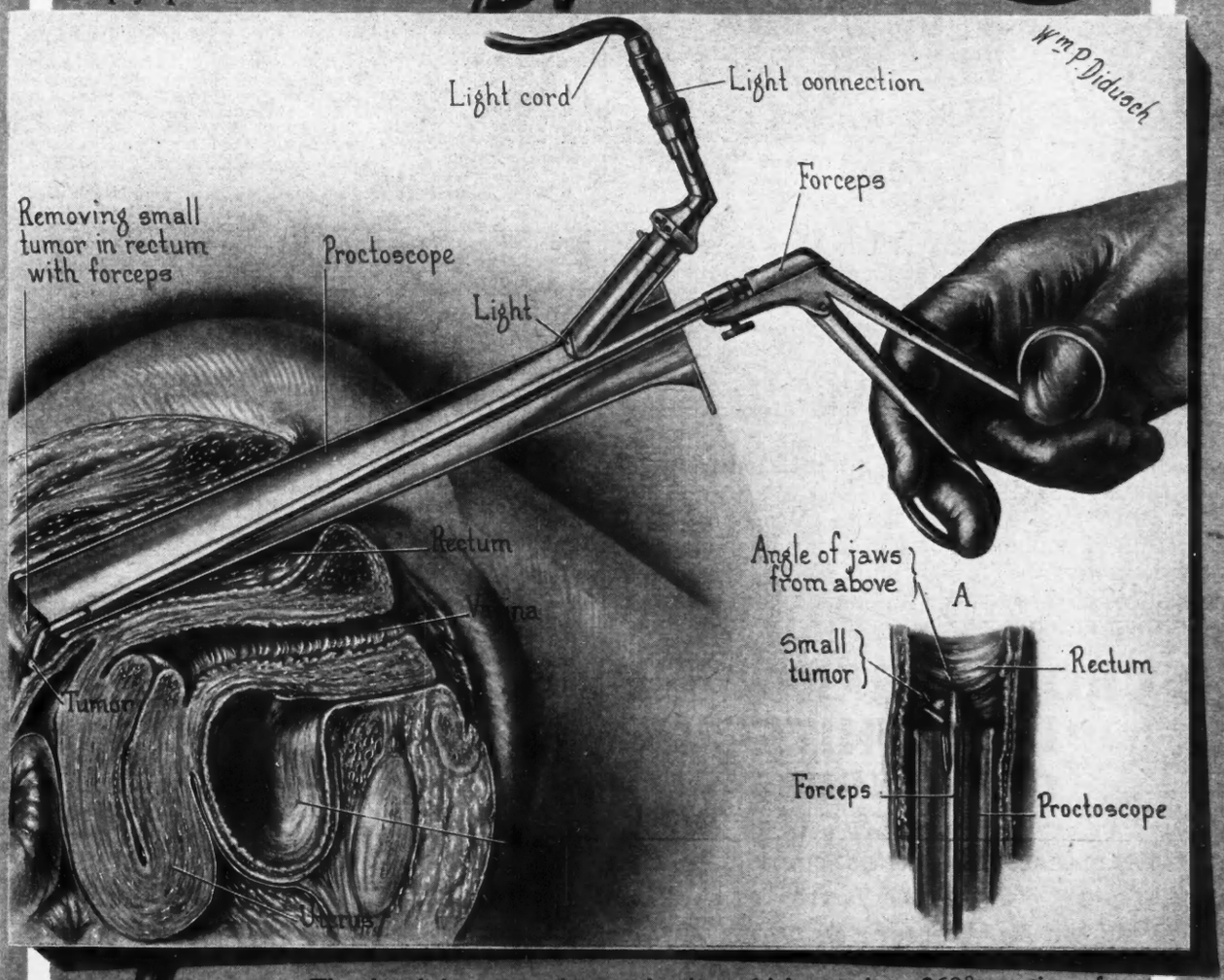

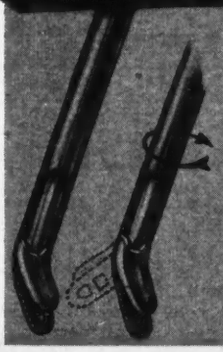

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Continued from Page 33

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